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ATHLETICS AND THE HEART: AN ELECTROCARDIOGRAPHIC AND RADIOLOGICAL STUDY OF THE RESPONSE OF THE HEALTHY AND DISEASED HEART TO EXERCISE[1]

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IN an appeal to the authorities, the conscientious inquirer into effects of violent exercise is bewildered by discrepancies and contradictions. [Abrahams.(1)]

The death of a competitor after an athletic contest is frequently followed by a flood of articles in the Press, the subject being "heart strain" or "athlete's heart". Excluding hyperpyrexia, the majority of sudden deaths associated with some feat of physical exertion occur on the golf links. (Abrahams.(1)) The few deaths that do occur in young subjects after exercise are not due to the effects of exertion on a normal heart, and are frequently due to hyperpyrexia. Death due to the stress upon a normal heart does not occur for the reason that an individual ceases to exert himself, owing to muscular or nervous fatigue, long before his heart is overstrained. (Schneider and Crampton.(35)) The symptoms of distress in an athlete bear little resemblance to those of heart failure. When athletic efforts stop because of physical exhaustion, there is rarely any cyanosis, venous engorgement, enlargement of the liver or cardiac œdema; any pain that is complained of is not of coronary type. The man who is in a state of collapse after exertion suggests a patient with hypoglycæmic shock or failure of the nervous system rather than cardio-vascular inadequacy.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on November 11, 1936.

Harvey,[43] in 1628, stated that "the more muscular and powerful men are, the stronger, thicker, denser and more fibrous their hearts". Since the seventeenth century the popular opinion has been that excessive physical exercise harmed the heart (Steinhaus[38]), and the physiological and clinical observations of the latter end of the nineteenth century seemed to confirm this opinion. Numerous German and other observers recorded dilatation of the heart during and after exercise, but these figures were based upon the estimation of heart size by palpation and percussion. (Williamson.[39])

Examination by the orthodiagraph and teleradiography fails to confirm this opinion, and Nicoli and Zuntz in 1914[29] found that while slight enlargement of the heart occurred during the progress of exercise, usually a diminution of heart size followed the exercise. This was confirmed by Gordon, Levine and Wilmaers[16] in 1924 in a group of marathon runners: the diminution of heart size after the race persisted in some men for a period up to twenty-four hours. There is no radiological evidence to suggest that any severe degree of dilatation can occur in the normal heart as a result of undue muscular exercise. (Kerley.[22])

During 1935 and 1936 a series of observations have been made upon the oarsmen of Melbourne University eight, the Ormond College crew, and the Scotch College crew. To attempt to draw conclusions would be to exceed the limits of justifiable deduction, and the results are presented, not as a statistical survey, but rather as examples of the response of the heart in healthy men.

Our procedure included the taking of X ray films at a tube distance of six feet; the men being in the upright posture, with the front of the chest against the cassette, the films were exposed during full inspiration. The position of full inspiration was chosen because of the alteration in heart silhouette with different positions of the diaphragm. (Roesler.[31]) In the earlier stages of this research the time of the exposures was short, but later exposures of two to three seconds were used and the outline of the heart on these films represents the maximum size of the heart during diastole.

Heart Size Before and After Exercise.

The men of the Ormond College crew were examined early in March, 1936, before serious training commenced, and the results may be taken as representative of the athletic type of university man in the untrained state. Films were taken of these men, and they were then exercised by running up and down stairs or "touching toes" until their pulse rate was over 100 beats per minute. A second film was then taken as soon as possible after cessation of exercise, the interval being in the neighbourhood of five to ten seconds.

The heart shadow was measured by means of the planimeter and estimates of the area of the heart as seen in postero-anterior films were obtained. Without exception the area of the heart was less after exertion than before exercise. The diminution in area varied between 1·7% to 13·2% of the original area before exercise, the mean reduction in area of the heart shadow being 6·3%. In every student immediately after exercise ceased, the transverse diameter of the heart decreased, the diminution lying between 0·2 and 2·3 centimetres. The conclusion drawn is that immediately after exercise the normal heart becomes smaller compared with its size at rest.

This has been confirmed by taking films of the heart actually while the student was pedalling a bicycle, and further films were taken immediately after cessation of pedalling. In every instance the heart was smaller after exercise than either before the work commenced or during the actual exertion. Films taken some minutes after exercise ceased showed that this contraction in heart size persisted for some time. In some oarsmen the heart was smaller three minutes after exercise ceased than immediately on cessation of pedalling.

From these two experiments we may conclude that the effect of exercise on the heart is to cause a reduction in diastolic area after the exercise ceases. Paterson[30] has shown that the reduction in area in the postero-anterior films seen above also occurs in lateral views of the heart. We therefore conclude that the volume of the heart in diastole is smaller after exercise than at rest before exercise.

The experiments upon the students pedalling a fixed bicycle against resistance were designed to show the changes in heart size actually during the performance of heavy work. These men pedalled until the pulse rate rose to 140 to 160 per minute. By strapping the man firmly to a vertical stand, postero-anterior films were taken at an exposure of two seconds; in most of these films the heart outline was clearly seen. The area of the heart could be obtained and the transverse diameter measured. In some students the heart decreased slightly in size during exercise, in three men it increased to a small extent, but the change in area was never very great. The greatest decrease in size was 4% of the original area, and the greatest increase was 5% of this area. These men were being exercised to a severe degree, and yet in no case was the dilatation during exercise of any magnitude. While it is admitted that the number of observations is small (seven men), it is felt that it is a reasonable conclusion that if dilatation of the heart does occur during exercise, the increase in volume is so small as to be within the limits of experimental error. This result agrees with the observations of Nicolai and Zuntz,[29] who found an average increase in transverse diameter of 0·4 centimetre during the performance of work and a decrease of 1·2 centimetres in transverse diameter of the heart immediately on the cessation of exercise. Gordon[15] found that the diminution in heart size persisted for some hours after the efforts had ceased.

We may therefore conclude that during exertion the normal heart does not dilate to any appreciable extent; immediately exercise ceases the diastolic volume of the heart becomes smaller than before exercise commenced and definitely smaller than during the actual performance of muscular work.

The explanation of this reduction in heart size immediately after the cessation of exercise is not easy. The size of the heart area as shown on the X ray film is merely a record of the volume of the diastolic

filling of the heart. The diastolic filling is largely controlled by the volume of venous blood flowing to the heart. The venous return to the heart is increased to an enormous extent by rapid muscular movement; when this movement ceases suddenly at the end of exercise, the muscles remain lax—the capillaries of the muscles remain widely dilated, and therefore the venous return to the heart diminishes suddenly. This sudden diminution in venous flow results in sudden diminution of diastolic filling of the heart, which is represented by a smaller shadow on the X ray film. As the splanchnic arterioles and capillaries return to the resting state after the exercise ceases, the venous return to the heart remains at a lower level. As the pulse rate is rapid for some time after exercise, the duration of diastole is short. The sum of these two factors results in a smaller heart shadow for some time after exercise ceases.

Dilatation of the heart can occur only when the rate of emptying of the heart fails to overtake the increased venous return during muscular work. In normal circumstances the increased frequency and output of the heart beat prevent any degree of dilatation.

> The most extensive venous return that can occur . . . appears to exert a load which is still within the physiological limits of response of the normal cardiac muscle. The final limiting factor in the normal circulation . . . appears to be not the heart but the peripheral vascular system. (Eyster.[42])

For purposes of comparison a number of out-patients with cardio-vascular lesions at Saint Vincent's Hospital have been investigated along lines similar to those used with the oarsmen. Patients with hyperpiesia, mitral stenosis, syphilitic aortic regurgitation, pulmonary fibrosis, chronic nephritis and thyreotoxicosis have been examined. Some of these patients had a mild grade of symptoms of cardiac failure, but none of them had severe congestive failure. X ray films were taken before and after exercise, graduated according to the individual limitations of the patient. In general the heart diminished in size after exercise in these patients, but the degree of reduction in area was smaller than in healthy students. In none of these patients did any degree of dilatation occur. These results are comparable with those of Williamson,[39] who found that about one-half of a series of abnormal hearts which were in good compensation responded to exercise, within their power, by a diminution in size.

Gordon and Strong[17] produced enlargement of the heart in rabbits by injections of spartein sulphate, which results in myocardial fibrosis and hypertrophy. These rabbits and normal controls were exercised in a treadmill to the point of collapse from physical exertion. Every animal, both normal and abnormal, had a smaller heart after exercise than before the exertion. In none of these rabbits did dilatation occur, even when muscular effort was pushed to the stage of collapse.

Therefore we conclude that during muscular work appreciable dilatation of the heart does not occur, and that immediately after the cessation of exercise the heart decreases in size.

Athletic Training and Heart Size.

The men of the Ormond College crew were reexamined at times when they were in full training. A comparison of the hearts before and after training is of some interest. Some of the men in the boat showed a greater degree of diminution in heart size after exercise when trained than before training commenced. In two men this change was very great.

In one man the heart area diminished by 5·5 square centimetres after exercise before he commenced training. After three and a half months' training, the heart area after the same exercise became smaller to the extent of 16·75 square centimetres.

In another man the area before training showed a 2% reduction on exercise; after training, there was a 9·3% diminution after exercise of the same grade.

Although it is tempting to suggest that the trained heart empties itself more efficiently than the untrained organ, this cannot be maintained. The heart shadow on the radiological film is a record of the diastolic filling of the heart. The smaller shadow of the trained heart after exercise is due to a greater falling off of venous return immediately after cessation of exercise. The process of athletic training concerns the nervous system, the lungs, the muscles and the skin, in addition to the cardio-vascular system as a whole, not merely the heart as a single organ.

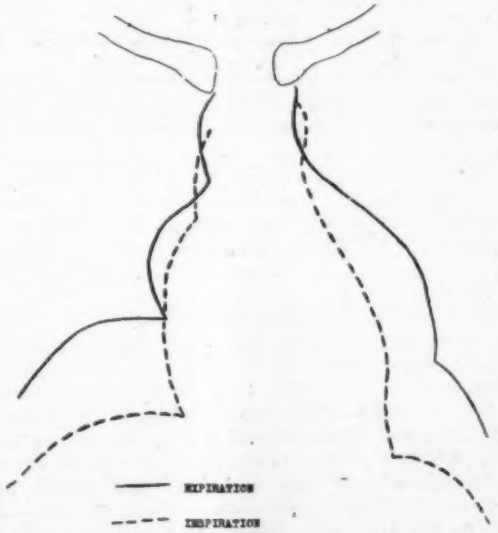

FIGURE I.
Change in shape of outline of heart according to different phase of respiration.

During exertion in the trained man the muscular and respiratory systems are working, not only at a more rapid rate, but also more efficiently than in the same individual in the untrained state. In Bainbridge's[2] opinion, during heavy work the action of the muscles in maintaining the circulation may be equal to or even transcend the importance of the heart. During exercise the arterioles in the tissues other than in the muscles and heart are constricted. This occurs to an extreme degree in the splanchnic bed. (McDowall.[41]) This mobilization of the

circulation for the performance of muscular movement comes into play immediately the trained man begins work. Immediately exertion stops the muscles relax.

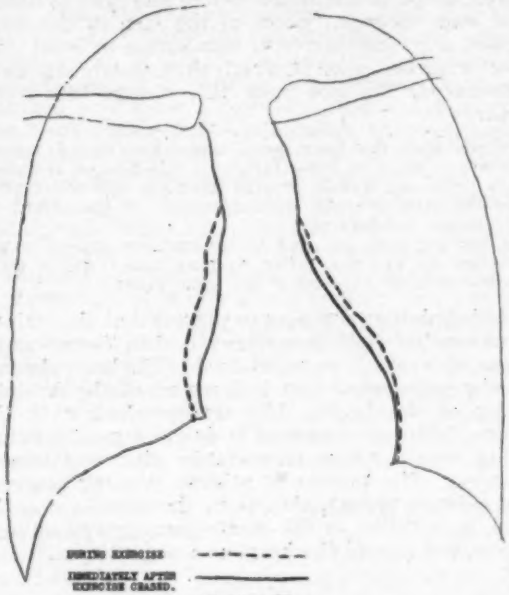

FIGURE II.
Decrease in area of heart on cessation of exercise.

Their capillaries are dilated more than in the untrained man, and the sudden fall in venous return to the heart is of greater magnitude in the trained athlete

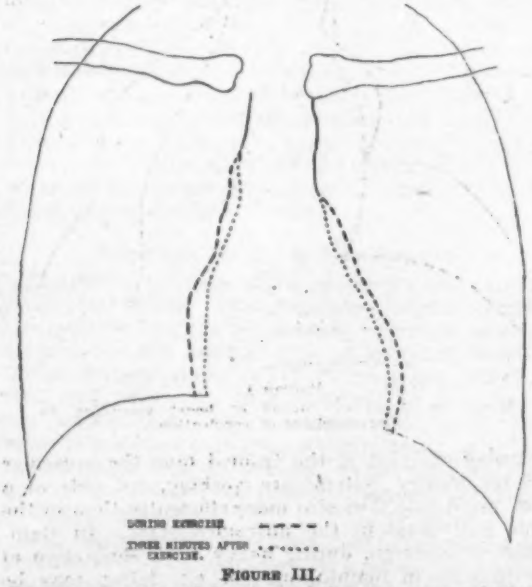

FIGURE III.
Decrease in area of heart persisting for some minutes after exercise ceases.

than in the untrained man. Therefore the diastolic filling of the heart falls off more rapidly in the trained than in the untrained man and the area of the heart on cessation of exercise becomes smaller in the trained athlete than before training commenced.

The change in size of the area of the heart shadow in the X ray film has been followed before and during training. In some men there is little change in the

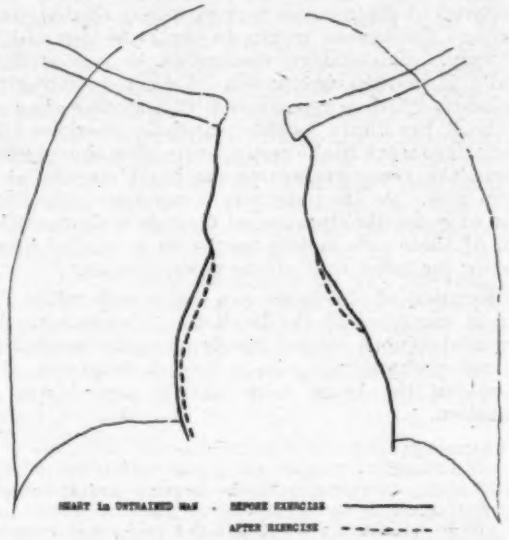

FIGURE IV.
Heart decreases in area after exercise even in untrained man.

size of the heart after some months of training, while in others the heart does appreciably increase in size with training. Those oarsmen in whom the heart

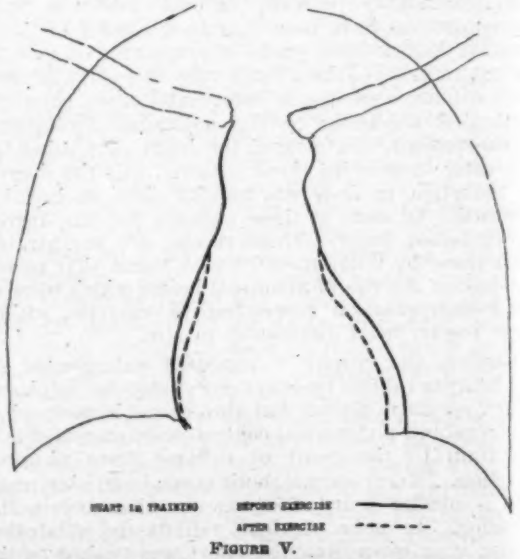

FIGURE V.
Decrease in area of heart after exercise is greater in the untrained man than in the same man (as Figure IV) before training commenced.

area at rest increased during training all showed a greater degree of diminution in heart size after exercise than before training. In each of these men

the size of the heart shadow after exercise was smaller in area when the man was trained than before training commenced. In physiological terms, immediately after exercise the diastolic filling of the heart in a trained athlete falls off to a greater degree than in the same man before training commenced.

The systolic output of the heart during rest is approximately the same at all times. The pulse of a trained athlete is slower during rest than in the same man before training began. Therefore the diastolic filling of the heart of an athlete at rest must be greater in volume and longer in duration than in the untrained man at rest. The larger volume of the heart during diastole means a larger X ray shadow. The trained athlete at rest has a relatively uneconomical cardio-vascular adjustment due to vagus over-action. (Steinhaus.[38]) The heart of an athlete at rest during training is in reality a dilated rather than an hypertrophied heart, compared with the same heart before and after training of the man.

The essence of training is a more rapid and more efficient mobilization of the whole vasomotor system to meet the demands of the muscles. The vagotonia of the trained athlete at rest is replaced by an extreme sympatheticotonic state immediately muscular exertion begins. Not only is the constriction of the splanchnic and other arterioles greater during exercise in the trained athlete than in the untrained man, but also the venous return is greater because the muscles of the trained athlete are working more efficiently during exercise. Immediately exercise ceases the trained vasomotor system returns rapidly to its resting phase. For these reasons there is a greater difference between the volumes of blood returning to the heart before exercise and after the termination of exercise in the trained athlete than in the untrained man. Therefore the difference between the areas of the heart at rest and of the heart after exercise is greater in the trained man than in the untrained individual.

In the period of the experiment it has been possible to obtain only a few films of men after they have ceased training.

In one man who rowed in the University boat in 1935 the heart area was 162·5 square centimetres when he was trained for a two-mile race. In March, 1936, when he had been out of training for some months his heart area had fallen to 148·2 square centimetres. He then went into training in the Ormond boat and at the time of the inter-collegiate races his heart area had increased again to the same size as when he was in training in 1935.

In this man the heart apparently enlarged during training and returned to normal after training ceased.

One oarsman who has rowed every year for twelve years was examined; no enlargement of the heart was detected by X ray examination and the heart contracted in area after exercise.

There is no evidence of permanent enlargement of the heart following training for athletics. (Gordon.[15])

This observation in oarsmen is contrary to the findings of Deutsch and Kauf,[10] who found that 27·3% of 150 oarsmen of an average age of twenty-four years had cardiac enlargement above normal limits.

Deutsch and Kauf,[10] at the Heart Station in Vienna, examined men and women engaged in amateur and professional athletics. Enlarged hearts were common in oarsmen, cyclists and ski-runners, while boxing, football and fencing hardly ever gave rise to cardiac enlargement. Even in those oarsmen who had large hearts, if the man ceased training the heart returned to normal size within a few months. (Deutsch.[11]) Christiansen, Krogh and Lindhard[7] observed three runners for a period of two years, and found an increase in the size of the heart which "adapts itself to increasing demands, but stops growing on constant demands".

The heart in wild animals is larger than the corresponding heart in domesticated animals of the same species. Clark[8] shows the ratio of the heart of wild duck to that of the tame duck to be 1·06 : 0·63, and greyhounds have a higher heart-body weight ratio than other dogs. The hare has a heart three times the size of a rabbit, and the hare's pulse, at rest, is only one-third of the rate of the rabbit's pulse. The heart of wild rabbits kept in captivity for six months became smaller than that of the ordinary wild rabbit. Puppies were exercised daily—others from the same litter were kept in inactivity; the ratio of the heart size of the active dogs to the sedentary dogs was 10 : 6. Finally, the heart of the thoroughbred racehorse is much larger than that of the ordinary hack. The heart of Phar Lap, in the National Museum at Canberra,[26] is larger than that of any other racehorse, and is equalled only by that of the English champion of the eighteenth century, Eclipse.

On the contrary, Dedichen,[9] found hypertrophy of the heart no more common in man in ski-runners than in labourers of the same age and build. Our observations, while few in number, would extend this opinion to include oarsmen, and we reach the conclusion that there is no radiological evidence of permanent enlargement of the heart following training for athletics.

Electrocardiographic Studies.

From a study of the shadows of radiology we pass to the even more indefinite fields of interpretation of electrocardiograms. Not only are the causes of variations in the electrocardiographic tracing doubtful, but we are not as yet certain of the source of origin of the normal physiological waves. For this reason the results of our study of the electrocardiograms of athletes' hearts are presented merely as examples of the variations that occur after physical effort.

The rise of blood temperature, the shortening of the systolic period, the even greater reduction in the diastolic pause and the variations in blood chemistry may all play some part in the changes occurring in the electrocardiogram during exertion. The position of the heart in the chest is also important (Herrmann[18]), particularly the displacement of the heart due to the increased diaphragmatic excursion after exercise.

The most striking feature of the electrocardiograms of athletic schoolboys and university students is the great amplitude of the deviations. The *T* wave in Lead I is often extremely high in athletes, as has

been noted by Flint[13] and others. This feature is common to both the trained and untrained man, but it must not be forgotten that even the untrained students examined were of the athlete type who had played games strenuously at school and university for many years. Even the schoolboys examined had been in training for at least two years, and were of a better physique than the average.

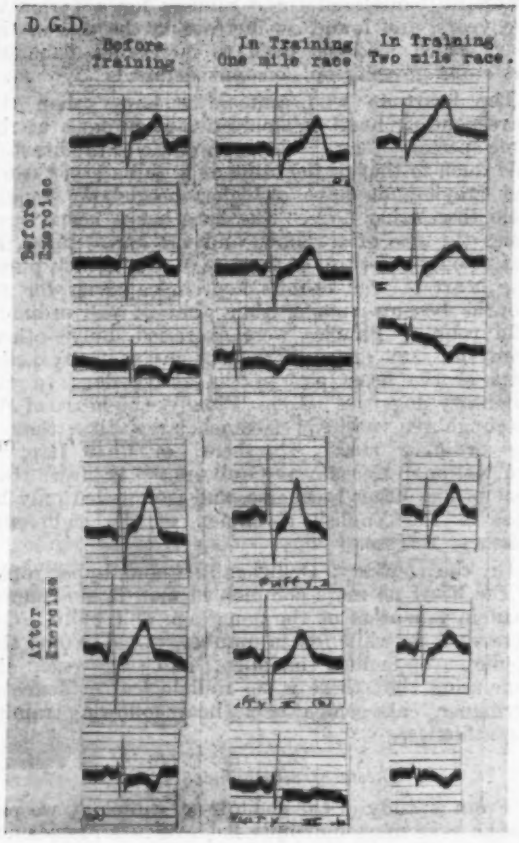

Figure VI.

The effects of training, as seen in the electrocardiograms, were not very great. Some of the men of the Ormond crew had electrocardiograms taken before training commenced and when they were prepared for a race. The time interval was approximately three to four months, and in this period the only striking change was an increase in the amplitude of the *T* wave in Lead I.

For example, in one man a deviation of T_1 of 0·2 millivolt in the untrained condition became one of 0·4 millivolt when trained for a mile race, and of 0·55 millivolt when trained for two miles. Six months after the last-mentioned electrocardiogram was taken, his *T* wave was small again. In the same man the *S* wave in Lead I became smaller during the period of training.

On the basis of the few men examined it is suggested that the electrocardiogram does change during training for athletic games, but that the changes are of small degree and are not permanent; the electrocardiogram returns to its previous form after training ceases.

The effects of severe exertion on the electrocardiogram were also recorded. The men of the University boat of 1935 were examined at the river before going out, and also immediately after doing a hard course of one mile. The pulse rate of those trained men before exercise was invariably slow, the most rapid being 62 per minute and the slowest pulse being 52 per minute. Immediately after a mile row the pulse rate was always over 100, and in some cases over 120 per minute. These pulse rates are of importance in interpreting changes in the electrocardiograms, for the shortening of the time of the heart cycle may have an effect on the form of the waves.

After rowing one mile the *P–R* interval was shortened and the *R–T* period was reduced to an even greater extent. In one man the *R–T* interval fell from 0·28 to 0·20 second. The shortening of the *P–R* and *R–T* periods was noted by Samaan.[33] The form of the complex also changed. The amplitude of the *P* wave increased, especially in Lead II, and the height of the *T* wave in Lead I sometimes increased; while in other men it was smaller after exercise. Similar changes in the electrocardiogram have been noted in French soldiers after a long route march. (Fredericq.[14]) An inverted *T* wave in Lead III is very common, but after exercise the depth of the deviation below the iso-electric line is usually increased. The height of the *R* wave in Lead II is usually greater after than before exercise.

The effects of prolonged strenuous exercise are shown also in a series of electrocardiograms taken before and after a hard game of squash.

In the two subjects examined the pulse rates after thirty minutes' playing were 124 and 140 respectively. In both these men the *P-R* interval was shortened by 0·02 second after exercise, while the *R-T* interval was reduced to an even greater extent.

This reduction of *R–T* period after exercise was noted by von Mentzingen.[28] The most striking change, however, was in the form of the complex. The amplitude of the *R* and *T* waves was greatly increased after exercise, the *T* wave in Lead II being twice as high after exertion as before playing.

The *S* wave in Leads I and II became deeper after exercise, and in one player a left preponderance present before the game was increased after thirty minutes of strenuous exercise.

There are appreciable changes in the electrocardiogram after even mild exercise of short duration, such as running up stairs or "touching toes" until the pulse rate rises above 100 per minute. The men of the Ormond crew had electrocardiograms taken before and after exercise of this type. While the time intervals of the complexes are not changed to the same extent as after the more strenuous grades of exertion, the form of the complex alters in the majority of men. Briefly stated, the *P* wave, the *R* wave and the *T* wave usually increase in amplitude, while the form of the *R–S–T* complex often changes. In some men in whom the origin of the *T* wave could

be described as a "high take-off" before exertion, the *T* wave tended to become more nearly normal after exercise. The "high take-off" of the *T* wave in athletes was noted by Bramwell and Ellis[5] in competitors in the Olympic Games in 1930.

To examine further the effects of exercise, an attempt was made to record the electrocardiogram of men while pedalling a stationary bicycle against the resistance of a brake. Tracings were taken before the subject started to pedal, during the pedalling, immediately after exercise ceased, and for some time afterwards.

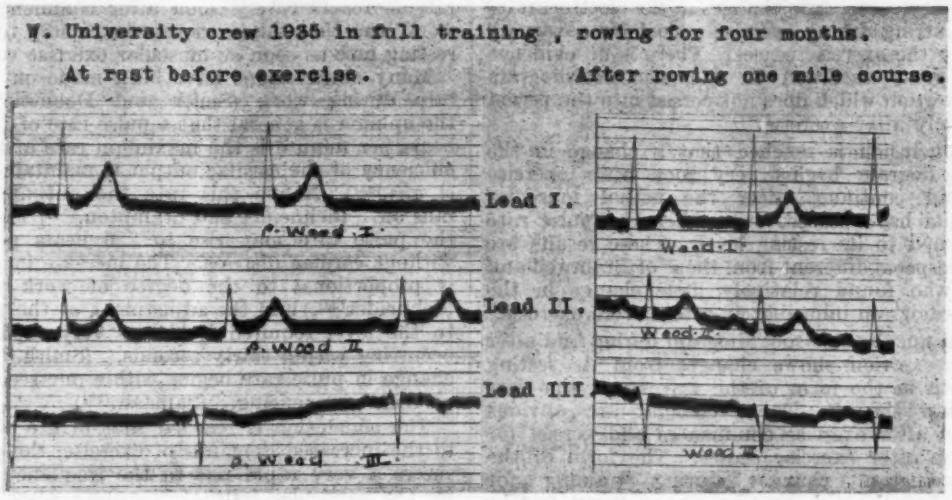

Figure VII.

Owing to movements of the lower limbs, the records in Leads II and III are distorted during the pedalling, but Lead I can be followed throughout the experiment.

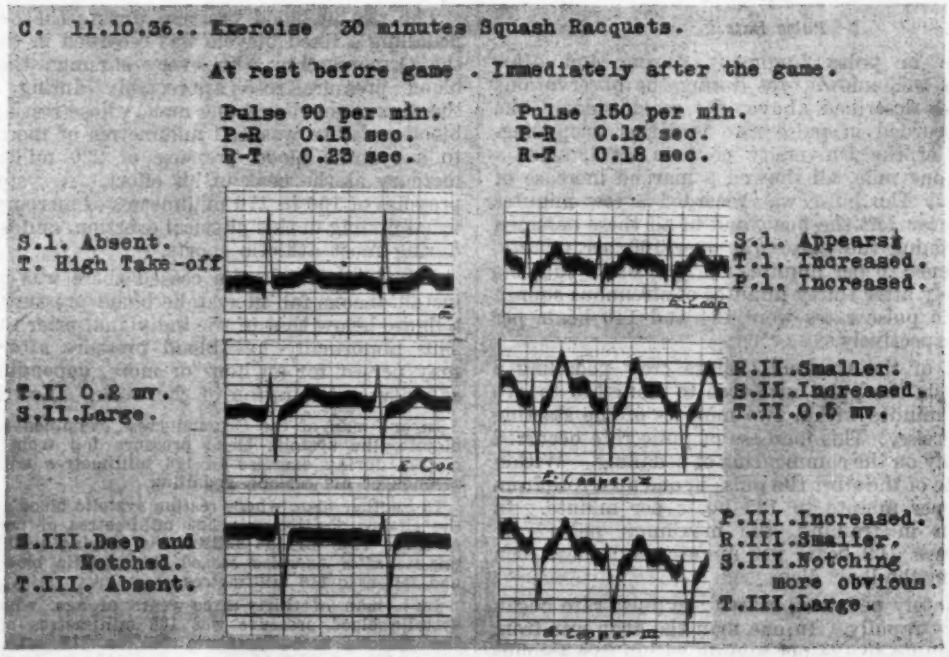

Figure VIII.

Even in Leads II and III a tracing can be obtained immediately exercise ceases.

Changes in the form of the *R–S–T* complexes begin

almost as soon as acceleration of the pulse, which in these trained athletes occurs very rapidly after the starting of work. The height of the *R* and *T* waves increases during the exercise, while the *P–R* and *R–T* intervals decrease. The changes in the electrocardiogram occurring during exercise persist for a varying time after the exercise ceases. There is no evidence of any gross variation in the electrocardiogram during exertion which does not persist into the period immediately after exercise.

The conclusion is reached that a change in the electrocardiogram begins very soon after exercise starts and continues after the actual muscular movements have ceased, even when the pulse rate has returned to its resting level. These results are in some respects different from those of Bramwell and Ellis,[5] who found relatively little change in the electrocardiogram immediately after severe exercise.

To sum up, the electrocardiogram during and after muscular exertion shows changes from its resting curve both in the form of the waves and the time relationship thereof. These changes are obvious very soon after exercise commences and persist for some time after exercise ceases. The form of the electrocardiogram changes during training for athletics, but these changes are not permanent. The changes in the electrocardiogram during exertion demonstrate that major variations from the accepted normal curve do not necessarily mean abnormalities of contraction of the heart muscle sufficient to interfere materially with the efficiency of the cardiac output.

Pulse Rate.

Increase in pulse frequency during and after exercise is well known, yet during the observations on athletes described above, the magnitude of the change recorded in pulse rate is rather surprising. The men of the University crew in 1935, after a course of one mile, all showed a marked increase of pulse rate. The pulse was recorded a few minutes after the crew left the boat, and in all these oarsmen in full training the pulse was over 100 per minute, and in some 120 per minute. In two other subjects immediately after thirty minutes of strenuous squash rackets the pulse rates were 124 and 140 beats per minute respectively.

In men of the Ormond College crew pedalling a fixed bicycle the pulse rate rose to from 140 to 170 beats per minute within two minutes of the starting of the exercise. This increase of pulse rate occurred very rapidly on the commencing of pedalling. Within one minute of the start the pulse in one man rose from 72 beats per minute to 132 beats per minute. In these men in training, the maximum pulse rate recorded was 168 beats per minute, and these men were not in the least distressed.

Immediately pedalling ceased the pulse rate began to fall very rapidly. In one man the drop was from 164 per minute to 124 per minute within five seconds of ceasing exercise. In another man the pulse dropped from 140 to 124 in the same period. Within one minute the pulse rate usually falls to within twenty beats of the rate before exercise, and within three minutes the pulse rate after this strenuous exercise was within ten beats per minute of the resting pulse.

These observations show that the heart accelerates immediately exercise begins, that the maximum heart rate is reached very soon after commencement of work, and that the heart returns rapidly toward the resting rate as soon as muscular exercise ceases.

Many observations have been made on the pulse rate during work (Smith and Doolittle[36]), and the opinion is general that a pulse rate of 160 to 170 beats per minute is the maximum rate at which the efficiency of the cardiac output is maintained. (Hill *et alii*.[21]) Above this rate the output per beat falls off. (Schneider and Crampton.[35]) In children the pulse rate may rise to 220 beats per minute without cardiac distress. The increase in pulse rate is proportional to the degree of work performed (Schneider[34],) and the adjustment of the pulse rate to the level required is always very rapid, usually occurring within thirty seconds. (Smith.[37]) Some change in pulse rate begins within one second of the exercise commencing. (Samaan.[33])

The conclusion is reached that in the adjustment to the increased demands of exercise, the pulse rate shows a very rapid rise to the frequency required, for the particular form of exertion; also, there is a rapid return toward normal once the exercise ceases.

The return of the pulse rate to normal after a standard form of exercise is one test used to determine physical fitness for athletics. (Billington.[4])

Blood Pressure.

The rise in blood pressure during the exertion of pedalling a fixed bicycle was recorded in the men of the Ormond crew. In every oarsman the systolic blood pressure rose appreciably during exercise; the maximum rise in one man, whose resting systolic blood pressure was 132 millimetres of mercury, was to a systolic blood pressure of 220 millimetres of mercury at the peak of his effort. A systolic blood pressure of 160 to 170 millimetres of mercury was the usual finding in this physical exertion, and is common during most athletic sports.

Immediately exercise ceased there was an almost instantaneous fall in systolic blood pressure, often to a figure below that of the individual prior to exercise. This abnormally low blood pressure after exercise may persist for an hour or more, depending on the severity and duration of the work. (Lowsley.[25])

In one man of the Ormond crew examined pedalling a bicycle, the systolic blood pressure fell from 220 millimetres during exercise to 176 millimetres within thirty seconds of his ceasing pedalling.

In another man, whose resting systolic blood pressure in the sitting position was 126 millimetres of mercury, the pressure rose to 164 millimetres during pedalling; one minute after exercise ceased the systolic blood pressure had fallen to 110 millimetres.

In a man of thirty-three years of age, whose resting systolic blood pressure was 136 millimetres of mercury, the systolic blood pressure fell to 116 millimetres of mercury after a hard game of squash.

The diastolic blood pressure rises during effort, but not to the same extent as does the systolic pressure; the result is an increased pulse pressure during exertion. After exercise ceases the diastolic

blood pressure and the pulse pressure both fall, usually to a figure below that recorded before the exercise commenced. (Lowsley.[25])

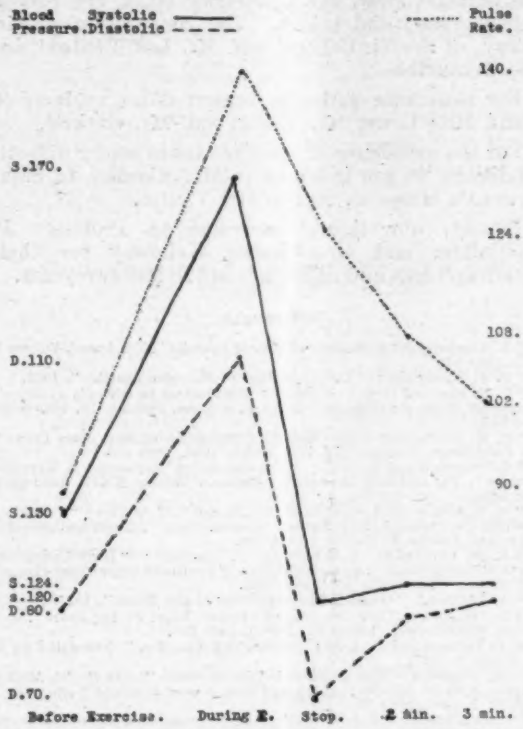

FIGURE IX.

G.McK., October 10, 1936, pedalling bicycle.

The conclusion is reached that extreme variations in blood pressure occur during exercise. The systolic blood pressure and the pulse pressure rise very soon after exercise commences, and fall immediately exercise ceases. The fall of blood pressure after exercise is often to a figure below that usual for the individual at rest.

Body Temperature.

A few observations have been made on the temperature reached after a strenuous game of squash. The maximum rectal temperature recorded after thirty minutes' exercise was 39·8° C. (103·8° F.). Other observations were 38·9° C. (102° F.), 39·3° C. (102·8° F.), 38·7° C. (101·8° F.). The resting rectal temperatures of the subjects examined was 37·2° C. (99° F.). This rise of body temperature persists for some hours after exercise ceases. In one series of observations the rectal temperature rose from a resting figure of 37·2° to 37·8° C. (99° to 100° F.) after a brisk walk. After thirty minutes' squash it rose to 39·4° C. (103° F.). Half an hour after the game it was still 38·9° C. (102° F.). Two hours later it was still 37·8° C. (100° F.). In the same subject after a walk of eight miles the rectal temperature rose to 38·7° C. (101·8° F.), one hour later it was still 37·6° C. (99·8° F.).

There appears to be no parallelism between the pulse rate and the body temperature. The pulse rate may return to normal, while the temperature is still markedly elevated. (Martin *et alii*.[27])

Other Changes during Exercise.

In the above observations the behaviour of the heart and circulation under the stress of physical effort has been recorded. The changes in the respiratory mechanism (Henderson[19]) and in the blood chemistry are of equal importance. (Hill.[20]) In this connexion it is of interest to note that one of the earliest observations on albuminuria after exercise was made by Sir Thomas Dunhill on the Ormond College crew of 1902. Dunhill[12] found that every member of the crew after a race passed albumin in the urine, while the urine was clear of protein when the men had not been rowing. There are other changes in the composition of the urine (Wilson *et alii*[40]); hæmaturia and glycosuria occasionally occur.

After prolonged strenuous exertion the blood sugar often falls to a very low figure. Best[3] has recorded blood sugar concentrations of 0·055 and 0·053 *per centum* in marathon runners at the Olympic Games in Amsterdam. Levine *et alii*,[24] also examining long distance runners, found that the fall of blood sugar in successful competitors was not very great and that these men finished in good condition. Men who were in a collapsed condition after the race had an extremely low blood sugar; one man brought in unconscious had a blood sugar of 0·045%. However, exhaustion can occur while the blood sugar is still normal (Cannon *et alii*[6]), and hypoglycæmia is only one of many factors concerned in fatigue. The nervous system, the neuro-muscular junction, and the muscle cell are all closely concerned with the establishment of fatigue during exertion.

Late Effects of Exercise.

Owing to the short duration of the experiments recorded in this paper, no conclusion can be drawn as to the late effects of exercise upon the normal hearts that we have examined. If enlargement of the heart occurred during training, this enlargement was due to excess diastolic filling rather than to muscular hypertrophy and the heart returned to normal size when exercise ceased. A few observations have been made on oarsmen who have been rowing for many years; in no case has the heart been larger than normal, and in the majority the heart is smaller than the average for the height and build of the individual. This is confirmed by the observations of Law[23] on the members of the Cambridge crews.

To investigate the late effects of athletics, the records of 100 men rowing for Ormond College between 1885 and 1905 were investigated. Of these men 24 are now dead, 17 from natural causes, seven from war injuries; 76 men are alive and well. The expected mortality of men of an average age of twenty-one years during the years in question is 31·8 (figures from the Australian Mutual Provident Society). The men who rowed for Ormond therefore show a lower mortality than the males of the general population.

These figures are similar to those obtained by Morgan in oarsmen at Oxford and Cambridge in the middle of the last century, and also by Meylen in Harvard crews (quoted by Law.[22])

We conclude this portion of our paper by a few quotations.

Bainbridge[1] writes:

> There is no direct evidence that, provided a man is physically sound and in good health, exercise however severe is ever harmful or followed by serious effects.

Sir Clifford Allbutt, quoted by Law,[22] wrote:

> The importance of muscular effort as a factor in cardiac injury has been much exaggerated.

Law[22] writes:

> No exercise however strenuous can permanently harm the healthy trained man.

Sir James Mackenzie writes:

> Personally never seen such a thing as a strained heart.

Sir Thomas Lewis writes:

> The circulatory organs are built to stand the strain which they themselves create. When the call is excessive it is the supply of blood to the muscles that will fail, not the heart.

We conclude with the motto on the title page of Harrington Sainsbury's book[23]: "*Ut pulsor, sic pulso.*"

Summary and Conclusions.

Twenty-five oarsmen between the ages of seventeen and twenty-five years have been investigated over a period of two years. The men have been examined before, during and after training for a severe effort. Observations have also been made on men playing squash rackets and pedalling a fixed bicycle against a brake.

Radiological investigations included films taken at six feet tube distance with two-second exposure in full inspiration. Electrocardiographic tracings, pulse rate, blood pressure and body temperature were also examined.

From these records and from a study of the literature available it is concluded:

1. During muscular exercise the heart does not dilate to any appreciable degree.

2. Immediately subsequent to exercise the heart is smaller than either before or during exertion.

3. If the heart enlarges during training, such enlargement is temporary and disappears when training ceases.

4. The size of the heart recorded on X ray films at two seconds' exposure is a representation of the degree of diastolic filling of the heart chambers.

5. Variations in the size of the heart during athletics are more likely to be alterations in the degree of diastolic filling of the chambers of the heart than to be due to thickening of the wall of the heart.

6. There is no evidence of permanent change in the heart following training for athletic sports.

7. Extreme physical effort in the trained athlete does not result in damage to the heart.

Acknowledgements.

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References.

(1) A. Abrahams: "Physiology of Violent Exercise", *The Lancet*, Volume I, 1928, page 429.
(2) F. A. Bainbridge: "The Physiology of Muscular Exercise", 1923.
(3) C. H. Best and R. C. Partridge: "Observations on Olympic Athletes", *Proceedings of the Royal Society*, Series B, London, Volume CV, 1929–1930, page 223.
(4) C. M. Billington: "The Medical Examination of Boat Race Crews", *The Practitioner*, Volume CXXXVI, March, 1936, page 310.
(5) C. Bramwell and R. Ellis: "The Circulatory Mechanism in Marathon Runners", *The Quarterly Journal of Medicine*, Volume XXIV, 1930–1931, page 329.
(6) W. B. Cannon, F. A. Campos, H. Lundin and T. T. Walker: "Conditions Affecting the Capacity for Prolonged Muscular Work", *American Journal of Physiology*, Volume LVII, 1928, page 680.
(7) E. H. Christensen, A. Krogh and J. Lindhard: "Investigations on Heavy Muscular Work", *Quarterly Bulletin of the Health Organization* (League of Nations), Volume III, 1934, page 388.
(8) A. J. Clark: "Comparative Physiology of the Heart", 1927.
(9) L. Dedichen: "The Influence of Physical Effort on the Heart", *Acta Medica Scandinavica*, Volume LIII, 1920, page 738.
(10) F. Deutsch and E. Kauf: "Heart and Athletics." Translated by L. M. Warfield, 1927.
(11) E. Deutsch: "The Heart and Athletic Strain", *The British Medical Journal*, Epitome 59, 1930. Abstracted from *Wiener Klinische Wochenschrift*, January 9, 1930, page 51.
(12) T. P. Dunhill and S. W. Patterson: "Albuminuria Following Severe Exercise in Healthy Persons", *Intercolonial Medical Journal*, Volume VII, 1902, page 334.
(13) H. L. Flint: "The Heart: Old and New Views", 1921.
(14) H. Fredericq: "*Traité de physiologie normale et pathologique*", Volume VI, 1927.
(15) B. Gordon: "The Effect of Effort on the Size of the Heart", *American Journal of Roentgenology and Radium Therapy*, Volume XIV, 1925, page 424.
(16) B. Gordon, S. A. Levine and A. Wilmaers: "Observations on a Group of Marathon Runners", *Archives of Internal Medicine*, Volume XXXIII, 1924, page 425.
(17) B. Gordon and G. F. Strong: "Studies on the Rabbit's Heart", *Archives of Internal Medicine*, Volume XXXII, 1923, page 517.
(18) G. R. Herrmann: "Electrocardiography and Cardiac Pathology from the Pathological Laboratory of the University of Michigan Medical School" (undated).
(19) Y. Henderson and H. W. Haggard: "The Maximum of Human Power and its Fuel", *American Journal of Physiology*, Volume LXXII, 1925, page 264.
(20) A. V. Hill: "The Physiological Basis of Athletic Records", "British Association for the Advancement of Science", Ninety-third meeting, Section I, Physiology, 1925, page 156.
(21) A. V. Hill, C. N. H. Long and H. Lupton: "Muscular Exercise, Lactic Acid and the Supply and Utilisation of Oxygen", *Proceedings of the Royal Society*, Series B, Volume XCVI, 1924, page 438.
(22) P. Kerley: "Recent Advances in Radiology", 1931, page 75.
(23) F. W. Law: "Some Clinical Aspects of Muscular Exercise", *The Quarterly Journal of Medicine*, Volume XXII, 1929, page 461.
(24) S. A. Levine, B. Gordon and C. L. Derick: "Some Changes in the Chemical Constituents of the Blood Following a Marathon Race", *The Journal of the American Medical Association*, Volume LXXXII, 1924, page 1778.
(25) O. S. Lowsley: "The Effects of Exercise on the Systolic, Diastolic and Pulse Pressures and on the Pulse Rate", *American Journal of Physiology*, Volume XXVII, 1910–1911, page 446.
(26) Sir Colin MacKenzie: Personal communication.
(27) E. G. Martin, C. M. Gruber and T. H. Lanman: "Body Temperature and Pulse Rate in Man after Muscular Exercise", *American Journal of Physiology*, Volume XXXV, 1914, page 211.
(28) A. von Mentzingen: "Shortening of *S*-Interval after Exercise", *Klinische Wochenschrift*, Volume XIII, 1934, page 88, abstracted in *The Journal of the American Medical Association*, Volume CII, 1934, page 972.
(29) G. F. Nicolai and N. Zuntz: "*Füllung und Entleerung des Herzens bei Ruhe und Arbeit*", *Berliner Klinische Wochenschrift*, 1914, page 821.
(30) R. Paterson and E. Paterson: "The Effect of Exercise on the Heart in Athletes", *American Journal of Roentgenology and Radium Therapy*, Volume XXXIV, 1935, page 158.
(31) H. Roesler: "Errors in Cardiovascular Roentgen Ray Interpretation", *Annals of Internal Medicine*, Volume X, 1936, page 299.
(32) H. Sainsbury: "The Heart as a Power Chamber", 1922.
(33) A. Samaan: "Muscular Work and Heart Rate", *Journal of Physiology*, Volume LXXXIII, 1934, page 313.
(34) E. C. Schneider: "Responses to Work on a Bicycle Ergometer", *American Journal of Physiology*, Volume XCVII, 1931, page 353.

(34) E. C. Schneider and C. B. Crampton: "The Cardiovascular Responses of Pre-adolescent Boys to Muscular Activity", *American Journal of Physiology*, Volume CXIV, 1935–1936, page 473.
(35) H. M. Smith and D. B. Doolittle: "Energy Expenditure during Horizontal Walking at Different Speeds", *Journal of Biological Chemistry*, Volume LXV, 1925, page 665.
(37) H. M. Smith: "Gaseous Exchange and Physiological Requirements for Level and Grade Walking", Carnegie Institute of Washington, Publication number 309.
(38) A. H. Steinhaus: "Chronic Effects of Exercise", *Physiological Reviews*, Volume XIII, 1933, page 103.
(39) C. S. Williamson: "The Effects of Exercise on the Normal and Pathological Heart", *The American Journal of the Medical Sciences*, Volume CXLIX, 1915, page 492.
(40) D. W. Wilson, W. L. Long, H. C. Thompson and S. Thurlow: "Changes in the Composition of the Urine after Muscular Exercise", *Journal of Biological Chemistry*, Volume LXV, 1925, page 755.
(41) R. J. S. McDowall: "The Control of the Circulation of the Blood", *Nature*, Volume CXXXVIII, Number 3490, 1936, page 488.
(42) J. A. E. Eyster: "The Clinical Aspects of Venous Pressure", 1929.
(43) William Harvey: "*Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus*", 1928.

THE LIFE HISTORY OF CYSTICERCUS BOVIS IN THE TISSUES OF THE OX.

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In the prevention of the spread of saginata tæniasis, meat inspection is important. For the guidance of the health officer and the meat inspector, the history of the cysticercus in the tissue of the ox is verbally and pictorially described.

Up to the time of Leuckart,[1] the connexion between *Tænia saginata* and *Cysticercus bovis* had been suspected but not proved. Leuckart actually produced *cysticercosis bovis* in calves by feeding them with ripe proglottides of *Tænia saginata*. He was thus able to study the life history of the cystic stage of this parasite. He observed a wide variation in the size of cysts of the same age. We have confirmed this variation and consider that the sizes of cysts of various ages given by Hertwig[2] are misleading, since he failed to recognize the great variations in size of cysts of the same age. In the earliest stages of development the embryo in the tissues of the ox is microscopic. Later, before the scolex is developed, it is a small spheroidal macroscopic body. Our observations in these early stages confirm substantially those of previous workers. In order to study the life history of *Cysticercus bovis* in detail we administered orally to each of thirty oxen 400,000 *Tænia saginata* eggs, and from time to time killed one or more of the animals and examined the cysticerci present. The details of the experiment were as set out below.

Materials.

The materials used in the experiment were: (i) forty-two uninfested oxen, of which sixteen were about ten months old, nineteen were about eighteen months old, and seven were about four years old; (ii) a batch of fresh *Tænia saginata* eggs, divided, after thorough mixing, into doses, each of 400,000 eggs. These eggs had been stored in physiological saline at 2° to 5° C. for two days after being collected from worms passed a few hours previously.

Description of the Experiment.

On January 19, 1934, 30 of the oxen were each given orally (that is, drenched with) 400,000 *Tænia saginata* eggs in water. The drenching was carried out by passing by the mouth low down into the gullet a rubber tube, through which the eggs, suspended in water, were administered. These 30 oxen comprised 12 about ten months old, 13 about eighteen months old, and 5 about four years old. The 12 remaining cattle were not given eggs and were kept as controls.

All the cattle, including the controls, were depastured, until slaughtered, on land not contaminated by sewage. The *post mortem* examinations of these animals were conducted over a period of two years. Five of the control animals were slaughtered after three to six months and no cysticerci were found. The heart, masticatory muscles, tongue and diaphragm of every animal were carefully examined by means of incisions 0·3 to 0·6 centimetre (one-eighth to one-quarter of an inch) apart. The dressed carcasses of the first 20 of the oxen slaughtered were completely cut up into similarly small slices. The remaining carcasses were examined by numerous incisions approximately 1·25 centimetres (half an inch) apart. Most of the organs of the first 13 cattle slaughtered were also carefully examined. The number of drenched animals slaughtered, together with the duration of infestation at the time of slaughter, is set out in Table I.

Table I.

The Number of Animals Slaughtered and the Duration of the Infestation.

Number of Drenched Animals Slaughtered.	Approximate Age when Experimentally Infested on January 19, 1934.	Age of Cysticerci when Oxen were Slaughtered.		
		Exact Age.		Approximate Age in Lunar Months. (Used for reference in text.)
	Months.	Weeks.	Days.	
2	10	4	0	1
1	10	8	4	2
5	18	12	0	3
	Years.			
5	4	12	0	3
	Months.			
1	10	17	3	4
1	10	20	5	5
1	10	25	5	6
2	10	28	5	7
1	10	32	3	8
1	10	36	2	9
1	10	40	4	10
1	10	51	4	13 (1 year)
3	18	65	5	16
2	18	79	4	20 (1¼ years)
3	18	109	0	27 (2 years)
30				

The main observations made at the *post mortem* examinations of the artificially infested cattle are summarized in Table II.

Table II should be examined in conjunction with the photographs of sections of heart muscle of the carcasses referred to herein.

Table II.

Observations on the Post Mortem Condition of Artificially Infested Cattle.

Approximate Age, in Months, of Cysticerci.	Ages of Oxen when Drenched.	Widest Diameter in Millimetres of—			Estimated Number of Measles in the Carcass.	Percentage of Cysts Alive.	Approximate Number of Live Cysts.
		Average-sized Measle.	Maximum-sized Measle.	Minimum-sized Measle.			
½ (15 days)[1]	Months. 10	3·0	6·5	0·7	3,600	Doubtful.	Doubtful.
1	10 10	5·0 4·0	14·0 13·0	4·0 3·7	30,000 24,000	Doubtful. Large.	Doubtful. Large.
1½ (6 weeks)[1]	12 12	5·0 6·0	10·0 12·0	3·5 3·0	7,500 4,500	99 20	7,500 900
2	10	4·0	14·0	2·0	27,000	50	13,500
3	18 18 18 18 18 Years. 4 4 4 4 4	4·0 7·0 7·0 7·0 6·0 7·0 7·0 6·0 7·0 7·0	7·0 11·0 11·0 11·0 12·0 14·0 14·0 12·0 10·0 13·0	1·5 3·0 3·0 3·0 4·0 3·0 3·0 2·0 3·0 4·0	13,800 15,500 11,400 17,200 8,600 30,000 14,800 19,700 17,200 15,200	58 33 61 } 51 48 68 1 2 16 } 5 4 0 } 24	8,000 5,100 6,900 8,200 5,800 300 290 3,100 680 0
4	Months. 10	8·0	14·0	6·0	4,500	0	0
5	10	6·0	7·0	2·0	18,000	25	4,500
6	10	6·0	10·0	5·0	20	10	2
7	10 10	1·5 2·0	6·0 7·0	1·0 1·0	9,000 12,000	0 0	0 0
8	10	2·0	8·0	1·0	6,000	0·03	2
9	10	4·0	6·0	1·5	3,000	0·03	1
10	10 (? older)	3·0	5·0	1·5	12,000	0	0
13 (1 year)	10	4·0	7·0	1·0	21,000	0	0
16	18 18 18	— 3·0 —	1·0 4·5 1·0	— 1·5 —	2 18,000 2	0 0 0	0 0 0
20 (1½ years)	18	— —	— 1·0	— —	0 Very few.	0 0	0 0
27 (2 years)	18	— — —	— — 1·5	— — —	0 0 6	0 0 0	0 0 0

[1] These cattle belonged to other experiments. They had only received 100,000 eggs each, whereas the rest had received 400,000 each.

Explanation of Table II.

Age.—The approximate age of cysticerci is put down to the nearest lunar month. For the exact age, refer to Table I.

Size.—The average size of the cysticerci was obtained in each carcass by measuring approximately 100 cysts taken at random. The average size was not always that of the mode, that is, the most frequent size represented. This was specially noted in some of the three-months-old cysticerci. Most of the cysts were either near the maximum or minimum size. At six weeks and at two months the

majority of cysts were of the average size. On first examining the three-months-old cysticerci, the impression is gained that they are considerably larger than those at six weeks or two months. However, the average size of three-months-old cysticerci was very little larger than that at six weeks. At three months there were certainly many more near the maximum size than there were at six weeks, but also there were many more near the minimum size. The maximum and minimum sizes of the cysts were obtained by thorough searching of the carcasses.

Number of Measles in Carcass.—We estimate that the most heavily infested of the carcasses of the first 13 animals slaughtered contained approximately 30,000 cysticerci. This estimatioon was arrived at after counting the number of cysts in small representative portions of the carcasses and estimating by weight what fractions these portions made of the total carcass. These estimates were only approximate.

The number of measles in the carcasses of the other experimental animals was, after complete slicing of the carcasses, estimated by comparison with the numbers found in the most heavily infested of the first 13 examined. In those carcasses in which only a small number was found, the cysts were counted.

Percentage of Live Cysts.—The percentage of live cysts in each carcass was estimated by examining a large number of cysts. One hundred to two hundred cysts would be examined closely in those carcasses in which the majority were alive. In those in which the majority were degenerated, many more were examined from all parts of the body. When no cysts were found alive, an enormous number in each carcass was examined.

Approximate Number of Live Cysts.—The number of live cysts was calculated, except when only a few were found, from the degree of infestation and the percentage of live cysts. This represents the risk of the beef if consumed raw after the cysts have attained maturity (probably six weeks).

The data in Table II represent first approximations only. The variations from animal to animal seen in the group of ten cattle killed three months after drenching suggest that the data recorded in respect of cysticerci of other ages do not represent all the possible variations that we should expect to find. However, it is hoped that this table, taken in conjunction with the photographs, will convey more to the reader than would any written description alone.

Remarks.

In this article the term "measle" denotes the cysticercus or bladder worm, together with its adventitious capsule.

The most important observations made in this experiment were the following:

1. The degree of infestation with cysts of the same age varied considerably from carcass to carcass, in spite of the great care taken in preparing the equal doses of eggs from the same batch and in administering them at the same time to the cattle. The number of cysts which developed from a dose of 400,000 "fresh" eggs we estimated to vary between 8,600 and 30,000. (See the details in Table II of the ten cattle slaughtered three months after receiving the eggs.)

2. The rate of development of bladder worms frequently varied considerably not only from animal to animal, but even in the same animal. For example, the majority of bladder worms four weeks old did not possess suckers; but occasionally one would be found with completely outlined, although small, suckers. The age of the individual measles could not be estimated accurately from size only. In this regard text-books quote Hertwig's figures, which are misleading. In the carcasses of our experimental animals occasional measles of one month old would be found measuring 13 millimetres to fourteen millimetres, equal to the maximum size we ever found at any age. In some of the infested animals the majority of the bladder worms were of a uniform size in spite of a wide diversity in size of the minority. The cysticerci, with or without their capsules, described by Hertwig, increased almost regularly in size with age over the period of observation (four to twenty-eight weeks), whereas we did not find this marked uniformity. We have not had access to Hertwig's original paper, and it is probable that the conditions of his and our experiments were substantially different. As previously mentioned, the size of bladder worms of the same age was also variable; but the variation was not as great as that of the measle, although substantial enough sometimes to make it impossible to determine the age of a bladder worm to within several months. For example, some three-months-old bladder worms appeared exactly similar to those of six and nine months. However, bladder worms of four, six and eight weeks could usually be identified as approximately of those ages.

3. We got the impression with measles of six weeks to three months old that the largest were degenerated; the largest living cysts were considerably smaller. Unless cysts after death temporarily swell, this observation seems difficult to explain.

4. Some measles were easily visible to the naked eye when only fifteen days old. Their undeveloped bladder worms, however, were microscopic.

5. Cysticerci were found degenerated at almost any size or age. From the age of six weeks onwards, an irregularly increasing percentage of cysts were degenerated.

6. Degenerated measles apparently decreased in size with age until they were finally completely absorbed. The rate of absorption varied greatly from animal to animal and from cyst to cyst in the same animal. In some animals practically all the cysts sixty-five weeks old had been completely absorbed, whereas one ox required two years to reach a similar stage. The residual cysts, although not a source of danger, disfigure the meat, and it is important to determine by a large experiment

when they completely disappear in the most refractory animals.

7. The length of life of cysticerci developed in the same animals at the same time varied greatly. The majority were dead at three months; but one cyst nine months old was found alive.

8. From six weeks to six months old the average size of measles did not vary much. The reasons are: (*a*) there is an increasing percentage of dead cysts with age (these decrease in size); (*b*) the cysts that live, on the average increase in size.

9. The maximum size did not vary much over the same period; but the proportion of measles closely approaching the maximum size varied greatly at different ages. The maximum size increased during the first three months and decreased from three months onward. Cysts at or near the maximum size at six weeks were usually dead. If alive, the contained bladder worms were relatively small. It was the thick capsule that really made for the large measurements.

10. After six months of age the average size of measles decreased. The reasons were that practically no cysticerci remained alive, many had been dead for some time, and none was found to exceed the maximum size of 14 millimetres.

11. On the average, cysticerci will live longer in eighteen-months-old oxen than they will in those four years old. (See Table II, three-months-old cysticerci.) Apparently the older animals develop immunity more quickly than the younger ones. This observation was repeatedly confirmed in other experiments.

12. Having in various experiments artificially infested with *Cysticercus bovis* oxen of ten months, eighteen months, three years, four years, and five years of age, I conclude that there is no marked natural age immunity.

I would reemphasize remark number 9. If sections (see illustrations) of the hearts of these animals are placed together for comparison, the most striking feature is the increase in number of large-sized cysts up to the third month, and the decrease in number of these large cysts thereafter.

The observations I have made may apply only under the particular conditions of the experiment described. With animals of different ages or with infesting doses of eggs of different sizes or ages, the results would probably be somewhat different. I have therefore confined my remarks to what were actually found, and have refrained from generalizations. To permit generalizations, much more experimental work would be necessary. On one occasion we found a live cyst which the circumstances of the case strongly suggested was at least fourteen months old. The ox in which this was found was four and a half years old and contained only two cysts, one of which was degenerated. If this live cyst were really fourteen months old or more, it must be looked upon as a most exceptional occurrence. It would also suggest that with a very light infestation of only two cysts the immunity developed was slight.

Macroscopic Study of the Development and Degeneration of Cysticercus Bovis.

Throughout the study on *Cysticercus bovis* I made observations on their macroscopic development and degeneration which appear to be of considerable value in assisting to assess the risk to consumers of infested carcasses. I have arbitrarily divided the life cycle of *Cysticercus bovis* in the tissues of the ox into six stages: three stages of development and three stages of degeneration. This has been done because at least six different appearances of *Cysticercus bovis* occur, and their recognition will be of value to the meat inspector and to those forming regulations which will adequately safeguard the consumers of beef and yet not be wasteful.

TABLE III.

The Description of the Contents of Measles at the Different Stages of Development.

Stage of Development.	Age of Cysticerci at which the Stage can Usually be Found.	Contents of Measle.
First	Weeks. 2–4	With the exception of a minute embryo, the entire contents are of a creamy consistency, yellow in colour.
Second ..	4–8	Living bladder worm, usually small, feebly developed, and opaque when very small. It is partly or completely surrounded by moist yellowish-green material of pasty consistency.
Third	6–12 (Range 1½–9 months.)	Contents consist solely of a complete bladder worm (bladder and scolex). The size and strength of the individual bladder worms increase with age until death occurs.

All cysts do not go through the three stages of development. Some die and degenerate when only the first or second stage is reached. The factor determining death is probably the immunity developed by the host.[3]

TABLE IV.

Description of the Contents of Measles at the Different Stages of Degeneration.

Stage of Degeneration.	Age of Measle at which the Stage can Usually be Found.	Contents of Measle.
First	Not many cysts have been seen at this stage, which must be of relatively short duration. Those that were seen were from six weeks to three months old.	Opaque (white) dead bladder worm; stiff, jelly-like rigidity of the caudal bladder.
Second ..	Six weeks to five months.	Moist pasty contents of a bright green colour (calcareous corpuscles recognizable microscopically). Bladder worm disintegrated.
Third	The large majority of cysts seven months old or older are in this stage.	Dry, dirty-yellow mass. Moderately hard, but can be broken into gritty material between the finger and thumb. The complete measle measures one to five millimetres in its widest diameter.

Remarks.

For practical purposes the division of cysticerci into the three stages of development and the three stages of degeneration is important, because I have noted, during the careful examination of a large number of cattle grazing on a sewage farm, that oxen that contained a cyst or cysts in the third stage of degeneration but none in the first or second stages of degeneration, contained no live cysts.

This observation suggests that only a relatively small number of cysts is required to produce immunity to further infestation under the natural conditions of a lightly contaminated sewage farm.

Summary.

1. An experiment, in which thirty oxen were artificially infested with *Cysticercus bovis*, to study its life history in the tissues of the ox up to a period of two years, is described in detail and illustrated by a series of photographs.

2. Under the conditions of the experiment it was shown that no cysticerci lived longer than nine months, and the majority were dead before they were four months old. The length of life of cysts varied greatly in the same or different animals.

3. The age of cysticerci over six to eight weeks usually cannot be estimated accurately.

4. Degenerated cysts gradually decreased in size with age until they were completely absorbed or in some instances left residual minute fibrous tissue scars. Absorption of all cysticerci was complete in two of three animals examined two years after they had been fed with a dose of eggs that was sufficient to produce a massive infestation. In the third animal almost all the cysts had been completely absorbed.

5. The macroscopic appearances of the contents of measles during their development and degeneration have been described in detail. The value of the description in assisting meat inspection has been pointed out.

Remarks on Photographs (Figures I to XIV).

The photographs shown in Figures I to XIV illustrate the experiment described on the life history of *Cysticercus bovis* in the tissues of the ox.

Except in the case of the photograph of the eight-months-old cysticerci, the cysticerci are shown as occurring in heart muscle. Figures IX, X, XI, XII and XIII, taken from a previous paper, are natural size. The others have been reduced.

It is impossible to illustrate adequately the results of the experiment by a photograph of only one section of the heart of each animal. Each photograph represents either the most or one of the most obviously infested portions of each animal, having regard to size and concentration of cysts. The appearance shown in the photograph of three-months-old cysticerci was typical of that of any section of the heart; whereas the photograph of cysticerci aged ten and thirteen months shows the most highly infested section of the heart that could be found. Many other sections at these stages showed no cysticerci.

Acknowledgement.

The general scheme of the experiment was outlined by Dr. W. J. Penfold, to whom my acknowledgements are due.

References.

(1) R. Leuckart: "The Parasites of Man", 1886.
(2) Hertwig: Quoted in Ostertag and Wilcox's "Handbook of Meat Inspection", 1916, page 423.
(3) W. J. Penfold, H. B. Penfold and M. Phillips: "Acquired Active Immunity in the Ox to Cysticercus Bovis", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, March 28, 1936, page 417.

RETENTION OF URINE AND THE USE OF URETHRAL CATHETERS.[1]

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I HAVE been asked by the Post-Graduate Committee to talk to you about retention of urine and the use of urethral catheters. I do not propose to elaborate on the differential diagnosis and treatment of the varying causes of urinary retention, but rather to approach the subject from the viewpoint of the general practitioner who, in an emergency, must decide on the provisional diagnosis and carry out the appropriate treatment. With this objective in mind, I shall tabulate the causes of urinary retention, whether "complete" or "partial", and say a few words on the immediate treatment of some of them. In addition, it is my intention to describe and to demonstrate a routine technique of urethral catheterization and bladder irrigation.

Retention of urine may be "acute" or "chronic". Whilst the former generally follows on the latter and is necessarily complete, the latter is usually partial and will include the varying amounts of residual urine that may be found. The most common causes of retention met with are as follow:

A. In Men.

1. Rupture of the bladder:
   - Traumatic.
   - Spontaneous.
2. Prostatic:
   - Malignant:
     - Adeno-carcinoma.
     - Scirrhous carcinoma.
     - Sarcoma.
   - Non-malignant:
     - Adenoma.
     - Fibro-adenoma.
   - Inflammatory:
     - Median bar.
     - Acute prostatitis.
     - Prostatic abscess.
     - Phlegmonous periprostatitis.
3. Urethral:
   - Stricture.
   - Rupture.
   - Calculous impaction and foreign bodies.
   - Periurethral abscess.
4. Phimosis and paraphimosis.
5. Neurogenic:
   - Cord injuries.
   - *Tabes dorsalis.*
   - Myelitis.
   - Post-operative state.
   - External trauma.

[1] Read at the New South Wales Post-Graduate Committee in Medicine General Revision Course, 1936.

B. In Infants and Young Children.
1. Valve formation in the posterior urethra.

C. In Women.
1. Chronic urethritis
2. Urethral stricture
3. Skenitis
(all three frequently associated.)
4. Foreign bodies in urethra.
5. Advanced genital prolapse.
6. Neurogenic, as in men; and occasionally
7. Myomata of the bladder neck.

Retention due to Prostatic Obstruction.—The diagnosis of retention due to prostatic obstruction is generally obvious after rectal examination. In some cases of partial retention with difficult micturition there may be no enlargement of the prostate palpable *per rectum*, and the final diagnosis may be arrived at only after cystoscopic examination.

For complete retention, the immediate treatment consists of urethral catheterization. The catheter should be tied in, and the bladder gradually emptied, six to eight ounces being removed for immediate relief, and six ounces being run out every hour. During this period fluids should be pushed by mouth.

The urine should be acidified, and kept acid, by a mixture such as the following:

Ammonium benzoate, 1·0 gramme (15 grains).
Glycerine, 2·0 cubic centimetres (half a drachm).
Chloroform water to 15·0 cubic centimetres (half an ounce).

This should be taken in water three times a day after food. Hexamine tablets in doses of 0·6 gramme (ten grains) should be given in conjunction.

The bladder should be irrigated once or twice a day, as will be described later, whilst the catheter is in position. The urgency of operation should be explained to the patient and admission to hospital arranged. If this is declined, or is impossible, the catheter should be left in position for three to four days. On its removal, voluntary micturition may become reestablished owing to the subsidence of the œdema, but the relief, if any, will be only temporary.

Regarding later treatment, it is only necessary to mention those available: prostatectomy, transurethral electro-resection and suprapubic cystotomy.

Prostatectomy is completely curative.

Transurethral electro-resection can guarantee only temporary relief, but, of course, may be repeated.

Suprapubic cystotomy is purely a symptomatic and palliative treatment, but may be demanded for a number of reasons, and may necessarily become permanent.

It is my firm belief that operation should always be advised when prostatic symptoms have become definitely manifest, and that the need becomes urgent when residual urine makes its appearance. Our mortality rate of under 3% at Lewisham would be capable of improvement if the public and profession in general would appreciate the insidious and inevitably progressive nature of the disease and the frequency of malignant changes and would seek expert advice at an early stage.

Acute Prostatitis and Prostatic Abscess.—Acute prostatitis and prostatic abscess are commonly Neisserian, but not infrequently non-specific. They may be post-influenzal. In the early stages complete rest in bed is necessary, and hot rectal douches and heat applied to the perineum may be useful. No urethral interference should be attempted during the acute stage. When pus is definitely present, the best treatment is hospitalization, transperineal incision of the prostate and drainage.

Phlegmonous Prostatitis.—Phlegmonous prostatitis is a not uncommon condition, and must be differentiated from malignant disease, especially from sarcoma in young adults. It is almost invariably non-specific, and is comparable to parametritis in the female. It very rarely tends to pus formation. Treatment consists of absolute rest in bed and the local application of heat, rectal douches, hot water bags and diathermy. This condition generally takes two to three months to clear up.

Urethral Valves.—Urethral valves may cause complete retention, and are the most common cause of partial chronic retention in children. They can be diagnosed only by cysto-urethroscopic inspection, and can generally be removed by the application of the high frequency current.

Urethral Stricture.—Urethral stricture commonly causes chronic retention, with distended bladder and dribbling overflow. Diagnosis from prostatic obstruction is made by the inability to pass moderate sized instruments. If a filiform bougie can be passed, the best treatment is internal urethrotomy and the regular passage of sounds. If a moderate sized sound (for example, size 6–9) can be passed, gradual dilatation (about two sizes) should be carried out at intervals of one week, and the regular passage of sounds instituted thereafter. If rapid contracture recurs, internal urethrotomy is palliative. Generally, old-standing or rapidly recontracting strictures are best treated by preliminary suprapubic cystotomy followed by external urethrotomy. This consists of the complete excision of the strictured area of the urethra and surrounding scar tissue, and of the reformation of the roof of the urethra, whilst the floor is left to reform itself. (Hamilton Russell operation.) Sounds should be passed at intervals for about twelve months.

Rupture of the Urethra.—Rupture of the urethra is best treated by immediate cystotomy and end to end anastomosis of the torn portions of the urethra.

Periurethral Abscess.—In periurethral abscess incision and drainage are frequently sufficient for immediate purposes. The urethral stricture which is generally present should be attended to subsequently.

Neurogenic Causes of Retention.—The commonest neurogenic causes of urinary retention are cord injuries and retention following operation. There is considerable controversy as to the most satisfactory treatment of the former. I am of the opinion that early suprapubic cystotomy offers the greatest hope of avoiding bladder infection and subsequent pyelonephritis, which is often the determining factor in this condition.

Retention Following Operation.—Post-operative retention of urine not infrequently responds to catheterization and the instillation of sterile glycerine into the bladder. It is extremely important that residual urine should be tested, especially when

post-operative catheterization has been resorted to, and even though apparently adequate quantities of urine are being passed. The retention of small quantities of urine is an exceedingly common cause of cystitis and pyelonephritis, complicating surgical interference apart from the urinary tract. A catheter should be passed night and morning until residual urine has completely disappeared.

In women, probably the most common causes of painful and difficult micturition and partial retention or urine are chronic urethritis, urethral stricture and skenitis. It is amazing how frequently one meets with this combination, and also how frequently an incorrect diagnosis has been made. The three conditions are generally found associated. Visual inspection of the external urethra, particularly during an attack, will reveal considerable redness and thickening and pouting of the lower lip of the urethra, and on palpation pus can usually be expressed from the orifices of the Skene's ducts. The treatment consists in the destruction of these ducts by means of the high frequency current. Cystoscopic inspection of the posterior urethra will reveal hypertrophied mucosa, or miniature polypi, which should also be destroyed with the high frequency current. Chronic inflammatory thickening and narrowing of the urethral lumen will, if present, require dilatation before a cystoscope can be inserted. Finally, massage of the urethra, either along the cystoscope or on a sound, should be performed.

Anuria.—Another subject which I should like only to mention before closing is anuria, which must enter into a differential diagnosis of urinary retention in a number of cases. As I have pointed out previously,[1] this condition is often essentially a surgical condition, and beyond bringing this fact once again before your notice, I do not propose to worry you with a further description.

The Technique of Urethral Catheterization.

It has been our practice for many years in this hospital to utilize a combined aseptic and antiseptic technique for all types of urethral instrumentation. This technique, in the main, can be adapted quite easily for use in general practice, and the adoption of such a routine will result in a considerable diminution of genito-urinary infections.

I make this statement after due consideration and forethought, as the number of patients referred with bladder, renal and genital infections, following catheterization in their homes, is quite appalling.

Another factor in reducing sepsis is the use of an indwelling catheter rather than frequently repeated catheterizations. The catheter should be replaced every three or four days, and should be allowed to drain only into some sterile vessel. Daily bladder lavage should be performed.

The following is the armamentarium necessary for urethral catheterization:

1. An all glass syringe.
2. A solution of oxycyanide of mercury which can be broken down for use to a strength of one in five thousand.
3. Sterile gauze (not cotton wool, as the latter leaves innumerable fibres which are extremely difficult to remove).
4. Methylated spirit.
5. Two pairs of plain dissecting forceps, one pair of artery forceps and one pair of scissors.
6. An antiseptic catheter lubricant, for example one composed of compound tragacanth powder, fifty grains; oxycyanide of mercury, two grains; glycerine, one ounce; and water to four ounces. This should be carried in a jar with a wide mouth.
7. Several rubber catheters—coudé and bicoudé.
8. A catheter guide or introducer, invaluable in many cases of difficult catheterization.
9. Adhesive plaster and tape.

The technique is as follows: The penis is well swabbed with methylated spirits and the prepuce, if present, is retracted and the glans is thoroughly cleansed. A solution of oxycyanide of mercury is repeatedly syringed into the anterior urethra, small quantities only being used at a time. No effort should be made to force the solution back into the posterior urethra or bladder. A small quantity of the lubricant can, if desired, be syringed into the urethra. The penis is held and well stretched with the fingers of the left hand. The catheter is picked up by artery forceps at the funnel end, and by dissecting forceps at the end to be introduced, and is thoroughly lubricated. It is passed to the bladder, the artery forceps being held between the little and ring fingers, and the dissecting forceps between the index finger and the thumb of the right hand. It will be found, by this manœuvre, that it is easily possible to refrain from handling the catheter in any way, and thus asepsis can be assured.

After the catheter has entered the bladder, it is withdrawn until the urine ceases to flow, and it is then again passed back into the bladder for about one inch. In this position, it will be found to be lying comfortably within the sphincter and not curled up in the bladder, a common fault, and will maintain a constantly empty bladder.

A piece of sterile tape is tied snugly around the catheter immediately external to the penis. The prepuce is drawn forward over the glans and is held in this position by a turn of adhesive plaster, to prevent the occurrence of paraphimosis, which may be a most disturbing complication. The tape is then draped along the penis and it too is firmly fastened down with adhesive plaster.

When there is evidence of chronic retention of urine, it is exceedingly important that not more than six ounces of urine should be withdrawn at any one time, and it is our practice to obtain slow decompression of the bladder by means of a hypodermic needle connected to the catheter. Fluids by mouth should be pushed to the limit of tolerance.

The number of patients who will not tolerate an inlying catheter is very small, and even this number may be reduced very greatly by explaining the importance of the procedure. It will generally be found that after the first twenty-four or forty-eight hours, very little discomfort of any sort is manifested.

When the bladder is completely empty, constant drainage is established by connecting the catheter

with a length of rubber tubing to a sterile bottle attached to the bedside. The end of the tubing should lie below the surface of some antiseptic solution in the bottle, to ensure a mild syphon action and to prevent air blockage. Bladder irrigations should now be commenced once or twice a day, depending upon the degree of infection of the urine. A weak solution of permanganate of potash, pale pink in colour, is run into the bladder through a funnel connected to the end of the catheter. This solution is washed backwards and forwards until the return is clear, and the remainder is completely removed with sterile water. Four ounces of a one in three thousand solution of silver nitrate in distilled water are injected and the catheter is clamped for fifteen to thirty minutes. The clamp is then removed and free drainage into the sterile bottle is reestablished.

We have found that the dirtier the bladder, the greater is the tolerance for silver nitrate, and the strength of this solution may be progressively increased up to one in fifteen hundred. It should at no time be strong enough to cause acute discomfort.

In conclusion, I cannot emphasize too strongly the importance of draining the catheter at all times into a sterile container, and of preventing the open end of the catheter from coming in contact with non-sterile articles. Should this be allowed to occur, infection is inevitable and elimination of the infection is impossible.

Reference.

(1) Richard G. S. Harris: "Surgical Anuria", The Medical Journal of Australia, August 10, 1935, page 173.

EPIDEMIC PLEURODYNIA.

By K. McK. Doig, M.B., B.S. (Melbourne),
Colac, Victoria.

In December, 1936, there occurred in this district several cases of an illness which appeared to be quite new as regards symptomatology. It was at first thought to be one of the many manifestations of influenza—a disease which is an ever-present refuge when a label is required for the complaint. The illness, however, was too clear cut a clinical entity to be dismissed so easily, and the extension of the epidemic throughout the district added interest to the problem from the point of view of diagnosis. Children were the first to be attacked. The illness came on suddenly, and was attended by severe pain in the chest. The respiratory rate was increased up to forty or more per minute, the temperature was raised to 38·9° C. (102° F.) or higher, and the pulse rate increased proportionately. At times the inspirations were accompanied by a catch as in pleurisy, and often there would be tenderness over the site of the pain. These alarming symptoms were, at first, thought to be due to an incipient pneumonia, but by the following day the child might be entirely free of symptoms or so much better that it was obvious that a serious prognosis was quite erroneous. In a number of cases the pain might return in a day or so, perhaps on the opposite side, but not so severely as in the initial attack. Generally, even in severe cases, the child was quite better in four days, and recovery was not attended by any weakness or prostration. Several children in the same family might be attacked one after the other. The following is an illustrative case.

On December 19, 1936, I.B., aged eight years, took ill in the afternoon with severe pain just below the right nipple. The temperature was 38·9° C. (102° F.). Respirations numbered forty in the minute. There was a catch in the breathing. The pulse rate was 110. No cough was present. Examination of the chest revealed no abnormality.

On December 20, 1936, the patient was much improved. The temperature was normal. The patient had no pain. The chest was still clear. The patient wanted to get up.

On December 21, 1936, slight pain was present in the region of the left scapula. The temperature was 37·2° C. (99° F.). The appetite was good and the patient still wanted to get up. There was no cough and the chest was clear.

On December 22, 1936, the patient had no pain. His temperature was normal. He was allowed up. His subsequent history was uneventful. No debility resulted.

The above is typical of the many moderately severe cases seen during the epidemic. Very mild cases occurred in which the child complained only for twenty-four hours and then recovered completely.

Later the epidemic spread to adults, and here the symptomatology became not quite so dramatic as in the children, although many did conform to the type seen in the younger patients. In adults there was a tendency for the pain to be more abdomino-thoracic than wholly thoracic. The pain was frequently referred to the epigastrium or the rib margins, to the right side more often than the left, but there was still, even when the abdominal type of pain predominated, the complaint that the breathing was embarrassed to a greater or lesser extent. Further, the disease appeared to be of longer duration in adults, and it mostly occurred in people before the age of thirty. A typical case history is as follows.

On January 22, 1937, J.K., aged twenty years, took ill in the morning with severe pain in the left side of the chest, just above the rib margin. His breathing was distressed, the respirations being 38 per minute. His temperature was 39·6° C. (103·4° F.). He had a cough which he thought was due to cigarettes. The leucocytes numbered 10,000 per cubic millimetre. Examination revealed no evidence of lung or pleural involvement.

On January 23, 1937, his condition was much improved. Pain was less and his temperature was falling. Chest examination revealed no abnormality.

On January 24, 1937, he complained of slight pain on the opposite side, but the initial pain had disappeared. He felt much better.

On January 25, 1937, his temperature was normal. All his pain had gone and he felt quite well.

In other cases occurring in adults, the pain was referred more to the region of the rib margins than in the case cited above, and the possibility of gall-bladder trouble or other abdominal conditions had to be considered. The absence of real rigidity or other serious signs or symptoms, the rapid improvement, along with the fact that perhaps other members of the family had been similarly attacked, served to establish the case as belonging to the same category as the others. Leucocyte counts were made in some of the cases, and these counts were generally in the

ILLUSTRATIONS TO THE ARTICLE BY Dr. H. BOYD PENFOLD.

Figure I.
Fifteen-days-old *Cysticercus bovis*. (Only 100,000 *Tænia saginata* eggs were given to this ox, whereas all the remainder received 400,000 each.)

Figure II.
Two-months-old *Cysticercus bovis*.

Figure III.
Three-months-old *Cysticercus bovis*.

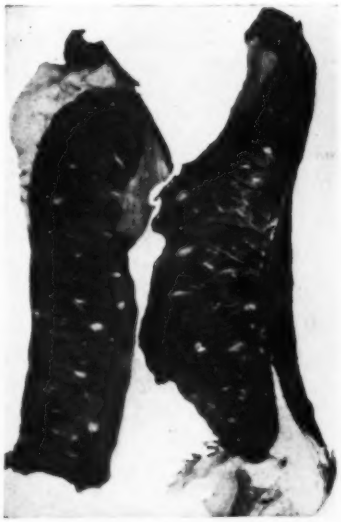

Figure IV.
Five-months-old *Cysticercus bovis*.

Figure V.
The remains of seven-months-old *Cysticercus bovis*.

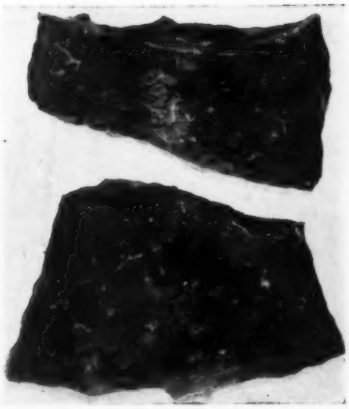

Figure VI.
The remains of eight-months-old *Cysticercus bovis*.

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Figure VII.
The remains of ten-months-old *Cysticercus bovis*.

Figure VIII.
One-year-old *Cysticercus bovis*.

Figure IX.
The remains of sixteen-months-old *Cysticercus bovis*.

Figure X.
The remains of sixteen-months-old *Cysticercus bovis*.

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FIGURE XI.
The remains of sixteen-months-old *Cysticercus bovis*.

FIGURE XII.
Complete absorption of cysticerci one and a half years after first being infested.

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Figure XIII.
Almost complete absorption of cysticerci one and a half years after first being infested.

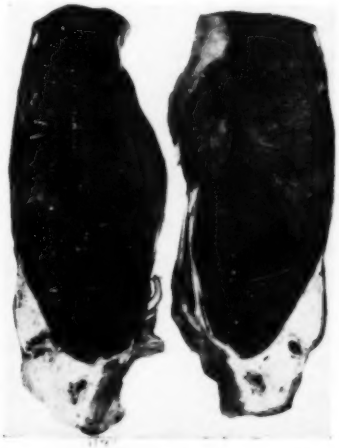

Figure XIV.
Complete absorption of cysticerci two years after first being infested.

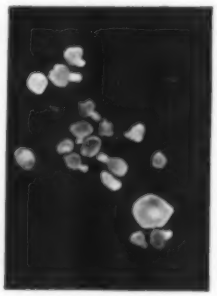

Figure XV.
Six-weeks-old bladder worms, all obtained from the same ox.

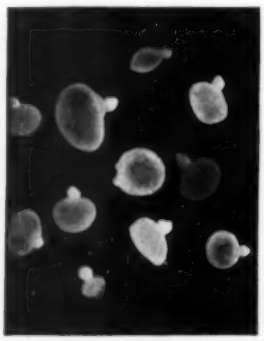

Figure XVI.
Three-months-old bladder worms, all obtained from the same ox. The variation in size is well presented.

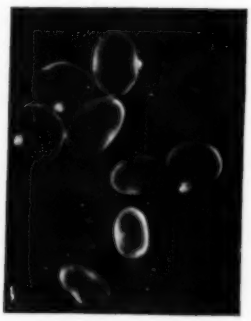

Figure XVII.
Five-months-old bladder worms. They represent the largest obtainable. No small ones are shown.

region of 10,000 or 12,000; and there was apparently nothing characteristic in the differential count. In one case there was a decided leucopenia, the count being 3,000. The patient was a girl of fifteen years, who had been ill for a week and who was admitted to hospital with a temperature of 39·7° C. (103·6° F.), but within twenty-four hours her temperature was normal and the girl very much better.

The outstanding features of the disease as exemplified in this epidemic are:

1. Its epidemic nature. The illness started in the town first and then spread to the surrounding district.
2. Children appeared to be first affected, and later young adults were attacked.
3. The onset with severe pain, thoracic in children, but either thoracic or abdomino-thoracic in adults, and along with the pain the respiratory embarrassment.
4. Absence of any physical signs of lung or pleural involvement.
5. Recurrence of pain, sometimes on the opposite side. When recurrence did take place, the pain was not so severe as that of the onset.
6. The good prognosis. No fatal cases occurred.
7. The complete recovery without complications or prostration.

There seems little doubt that the outbreak was one of epidemic pleurodynia, which, so far, has been reported only in America. J. C. Small, writing in Cecil's "Textbook of Medicine", states that the disease was first described by Dabney in 1888, when it occurred in Virginia. It occurred in the same locality again in 1923, in New York in 1923, and in Pennsylvania and New Jersey in 1924.

Robert G. Torrey refers to the disease as epidemic diaphragmatic pleurodynia or "devil's grip", and describes cases that occurred in Philadelphia in 1924. In his description he states that the onset was often accompanied by a chill, and that sweating was a feature of the disease. These features have not been noted in the present epidemic. Torrey is of opinion that the pain is due to involvement of the diaphragm or the diaphragmatic pleura.

In view of (*a*) the absence of leucocytosis, (*b*) the decline of the fever with cyclic recurrences, (*c*) the absence of inflammatory foci, and (*d*) the increase of the eosinophile cells during convalescence, Small thought that the infecting agent might be something in the nature of a protozoan organism, and not a bacterium. He examined smears of blood from patients suffering from the disease, and has described a plasmodium occurring within the erythrocyte. He used Wright's stain, and says that the plasmodium stained a bright blue. At the same time he is of opinion that his findings should be taken as more or less suggestive of the cause of illness and would like confirmatory evidence as to the presence of the plasmodium. Blood films taken in the present epidemic have not demonstrated the plasmodium, but this may be due to inexperience in the recognition of the parasite.

In view of Small's conception of the ætiology of the disease, quinine has been given, but whether this drug is an absolute necessity is open to doubt, for the present outbreak responded very well to rest in bed and the exhibition of simple sedatives.

It is possible that another epidemic may occur next summer, when, perhaps, further light may be shed on the disease, and better technique and experience be available in an endeavour to confirm the findings of Small.

Conclusion.

A disease of an epidemic nature has been described, which in its symptomatology and course appears to be identical with that which has been described in America as epidemic pleurodynia.

Bibliography.

J. C. Small: "Textbook of Medicine", by Cecil, "Epidemic Pleurodynia".
J. C. Small: "A Protozoon Organism within the Erythrocytes of Patients Suffering from Epidemic Pleurodynia ('Devil's Grip')", *The American Journal of the Medical Sciences*, Volume CLXVIII, 1924, page 270.
Robert G. Torrey: "Epidemic Diaphragmatic Pleurodynia or 'Devil's Grip'", *The American Journal of the Medical Sciences*, Volume CLXVIII, 1924, page 564.

TRIGEMINAL NEURALGIA AND DISSEMINATED SCLEROSIS, WITH A REPORT OF A CASE.

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Although trigeminal neuralgia in association with disseminated sclerosis has been noted by several observers, it is doubtful if this combination of conditions has received full recognition as a true clinical entity. Thus Frazier,[1] who has had an enormous experience with trigeminal neuralgia, states as recently as 1934 that he cannot recall any case of true major neuralgia in a patient with disseminated sclerosis. On the other hand, Harris[2] has encountered 41 patients whose chronic spastic paraplegia was complicated by trigeminal tic; and of these, seven had bilateral tic. Parker[3] also describes in detail four similar cases, the tic being bilateral in one. A further bilateral case is described below.

The neuralgia in these cases corresponded to true trigeminal tic in all particulars such as type and severity of pain, remissions, trigger zones and reaction to treatment. Almost always the neuralgia developed later than the signs of spinal disease. In a few instances amongst Harris's patients the influence of heredity was apparent, one generation having tic and the next disseminated sclerosis. The sexes appear to be equally affected.

A most careful *post mortem* study was made of one of the cases described by Parker. His patient had left-sided tic and died four days after operation. The brain, spinal cord and both trigeminal nerves and Gasserian ganglia were examined. Parker summarizes the necropsy findings as follows:

> The disease of the central nervous system from which the patient suffered was multiple sclerosis. Innumerable plaques were scattered throughout the brain and spinal cord. Well-marked and relatively old plaques were found on the main sensory nucleus of the right fifth nerve. On the right side and on the lowest part of the spinal nucleus and root was a plaque. The right posterior root was normal, but on the left side, at the point of emergence of the root from the pons and for a short distance along

its extrapontine course, there were two relatively old sclerotic plaques. The Gasserian ganglia and the central portions of the peripheral nerve trunks were free from any sclerotic process.

The source of the pain remains obscure. Harris, who thinks that the pathological process in trigeminal tic is a terminal neuritis of dental nerve filaments, considers it not surprising that patients with disseminated sclerosis should have bilateral tic, if in addition to the neuritis there are plaques adjacent to or invading the spinal roots of the fifth nerves.

Whatever may be the cause of the pain, there seems little doubt that disseminated sclerosis in association with unilateral or bilateral trigeminal neuralgia is a definite clinical entity, and that the neuralgia responds to ordinary surgical measures. There is no need to debate here whether the surgical measure should be injection of the ganglion with alcohol or division of the posterior root. In bilateral cases, however, it is of the utmost importance to adopt the procedure that is the more likely to spare the motor root, for it would be a most unfortunate matter for a patient to be left with a permanent bilateral paralysis of the muscles of mastication. In the case reported below, open operation was resorted to.

Case Report.

Mrs. C., aged forty-nine years, was first seen on July 15, 1936, when she complained of severe neuralgia on both sides of the face and stiffness of the left arm and leg. Her story was that as a young woman she had noticed a little difficulty in pulling the left leg round when performing the circular waltz. She had been aware of little more until five years ago, when the left leg became stiff and weak, with numbness and a feeling of pins and needles below the knee, and coldness and blueness of the foot. Two years later the left arm began to be similarly affected. Soon afterwards, that is, about three years ago, she had her first attack of facial pain. This was on the right side and was paroxysmal. Sometimes she would be free of it for several weeks and then would have repeated bouts, lasting ten to fifteen minutes, over a period of several days. The pain would occur both in the upper and lower jaw, and would be initiated by touching the gum, opening the mouth, taking a drink, washing *et cetera*. Six months ago the pain began to occur on the left side also. Her condition now became desperate, for she was practically never free from pain on one side of the face or the other. At times both sides were affected simultaneously. According to her own statement, she would go without food or sleep for a week at a time. Many drugs had been ordered her, but she had had no real relief. Micturition was frequent and precipitant. She had had a stillborn child eight years previously and a hysterectomy three years ago.

The patient looked care-worn and haggard. She was edentulous. There was some weakness of the left facial muscles, but otherwise the cranial nerves, including the fifth, were normal to examination. There was no nystagmus. The left upper and lower limbs were weak, stiff, cold and blue. The left leg was œdematous also. There was no ataxia or intention tremor. The deep reflexes were all very active, except the ankle jerks, which were diminished. The superficial abdominal reflexes were absent and both plantar reflexes were extensor. There was a little patchy alteration in sensation.

On August 18, 1936, the left Gasserian ganglion was exposed and the sensory root was partially divided and avulsed. Care was taken to preserve the motor root and also the ophthalmic fibres in the sensory root. Complete anæsthesia of the area supplied by the mandibular division of the nerve was obtained, but only partial anæsthesia of that supplied by the maxillary division. Evidently the division of the sensory root was insufficiently bold.

Ten days later the right sensory root was similarly divided, and on this side complete anæsthesia of the areas supplied by the maxillary and mandibular divisions was obtained. The patient experienced no eye trouble and no disability in eating, other than that ordinarily encountered from the anæsthesia obtained. She left hospital a fortnight later.

During the six months that have elapsed since operation there has been no pain of any kind. In view of the incomplete division of the left maxillary fibres it is possible that pain may eventually recur. The patient has a voracious appetite and enjoys life in spite of the severe disabilities she still has.

Acknowledgement.

I wish to express my great indebtedness to Dr. E. Graeme Robertson for the diagnosis in this case, the advice to operate, and the references to the literature.

References.

(1) C. H. Frazier "Bilateral Trigeminal Neuralgia", *Annals of Surgery*, Volume C, 1934, page 770.
(2) Wilfred Harris: "Bilateral Trigeminal Tic", *Annals of Surgery*, Volume CIII, 1936, page 161.
(3) H. L. Parker: "Trigeminal Neuralgic Pain Associated with Multiple Sclerosis", *Brain*, Volume LI, 1928, page 46.

Reports of Cases.

A CASE OF TETANUS.

By R. N. Burton, L.R.C.P. and S. (Edinburgh), L.F.P. and S. (Glasgow),

Texas, Queensland.

C.P., a care-free youth of eighteen, was admitted to the Texas District Hospital on August 3, 1936, with a sinus in the right ischio-rectal region, resulting from a laceration by a petrol tin on July 18. The tin was stated to have been clean. No antitetanic serum was given. The sinus, which was about one and three-quarter inches long, was opened freely on the following day and a gauze drain was inserted. While under the anæsthetic the patient had a violent and prolonged convulsion, the cause of which was a mystery until explained by subsequent events. On the morning of August 6 he remarked that his leg felt stiff, but the significance of this was not recognized at the time. It transpired later that he first noticed stiffness in the leg on the day before admission to hospital. At about midday on August 6 he had the first spasm. These became more frequent and severe during the rest of the day and night, but he made no complaint whatever until 9 a.m. on August 7, when he complained of epigastric pain.

On examination the abdominal muscles were very rigid; and masseteric spasm made the diagnosis plain. He was at once given 12,000 units of antitetanic serum intramuscularly, followed by "Avertin", 4·8 cubic centimetres (80 minims), by rectum. Four hours later another 13,000 units of antitetanic serum were given intramuscularly. Two hundred thousand units arrived by aeroplane at 2.30 p.m. and were given intravenously under ether anæsthesia. Convulsions again occurred, and as soon as possible "Avertin", 6·42 cubic centimetres (107 minims), was given. No further serum was administered throughout the illness. An hour later the wound was curetted and swabbed out with pure peroxide of hydrogen.

Spasms, which quickly became severe and frequent, were controlled by "Avertin" in doses of 4·8 or 6·4 cubic centimetres until 9.30 p.m. on August 9, when he was unable to retain any more. Morphine, 0·015 gramme (one-

quarter of a grain), with atropine, 0·32 milligramme (one two-hundredth of a grain), gave a respite of eight hours, and then "Amytal", 0·54 gramme (nine grains), was given by the rectum, and later paraldehyde, 24 cubic centimetres (six fluid drachms), were given by the rectum with good effect. After the second dose in each case restlessness and noisiness supervened, becoming extreme after the third dose; both were then stopped and administration of "Avertin" was resumed. The following sedatives were also tried, namely, bromide of potash and chloral hydrate, of each 0·6 gramme (ten grains), and "Sodium amytal", 0·18 gramme (three grains), orally, the former causing a very irritating rash and the latter hallucinations; "Luminal", 0·18 gramme (three grains), was also given hypodermically. All were found to be much inferior to "Avertin", although after August 13 they were able to control the spasms, which were then less severe and eventually ceased on August 16.

Vigorous local treatment, consisting of swabbing the wound every two hours with equal parts of peroxide of hydrogen and water, was continued throughout to prevent further absorption of toxin. All dressings and handling were done under "Avertin" whenever possible. Masseteric and abdominal rigidity ceased on about August 22, but stiffness in the right leg and foot remained for another week. Knee jerks and plantar response were present throughout.

Comment.

In regard to dosage of antitoxin, Cole's[1] technique, based on Spooner's work, was followed, namely, a single large intravenous dose of 200,000 units and no local treatment for at least an hour afterwards. Some American observers advise 100,000 units only, and claim that further doses are merely wasted. A further pronouncement on this question would be welcome, as the expense is heavy.

This case appears to confirm Cole's view that the prognosis is directly proportional to the length of the incubation period and what he calls the period of onset; these were sixteen and four days respectively, which made the prognosis appear fairly good. Nevertheless the spasms were severe and would apparently have quickly gone on to the stage of opisthotonos but for treatment.

The following list gives the average periods of freedom from spasms following the drugs used, from August 7 to 11 inclusive, when spasms were most severe.

"Avertin", 6·42 cubic centimetres (107 minims)	10½ hours
"Avertin", 4·8 cubic centimetres (80 minims)	6½ hours
"Amytal", 0·54 gramme (9 grains), *per rectum*	5½ hours
Morphine, 0·015 gramme (one-quarter of a grain), and atropine, 0·32 milligramme (one two-hundredth of a grain)	3½ hours.
Paraldehyde, 24 cubic centimetres (six fluid drachms), *per rectum*	3½ hours
"Luminal", 0·24 gramme (four grains), *per rectum*	2½ hours
Bromide of potash and chloral hydrate, of each 0·6 gramme (ten grains)	1½ hours

"Avertin", 6·42 cubic centimetres (107 minims), was the basal anæsthetic dose, that is, 0·1 gramme per kilogram of body weight. There appeared to be no advantage gained by giving the smaller dose. A total of 54 cubic centimetres (one ounce six drachms) of pure "Avertin" was given over a period of six days. Spasms ceased in from three to five minutes of administration. Cole states that "Avertin" is especially useful in relaxing masseteric spasm. In this case the patient was able to take food freely by the mouth. The writer began treatment with "Avertin" with some confidence, having used it for basal anæsthesia on all major operation cases since 1931, and never exceeding 0·1 gramme per kilogram of body weight. It has never given a moment's anxiety.

The resemblance of some cases of tetanus to those of an acute abdominal condition, as mentioned by Paterson,[2] was very noticeable on August 7 in this case.

References.

[1] L. Cole: "The Treatment of Tetanus", *The British Medical Journal*, June 13, 1936, page 1191.

[2] A. E. Paterson: "Tetanus: Its Diagnosis and Treatment, with a Summary of Twenty-Six Consecutive Cases", THE MEDICAL JOURNAL OF AUSTRALIA, June 28, 1930, page 832.

Reviews.

THE THYREOID AND ITS DISEASES.

DURING the last few years many excellent books have been published on the surgery of the thyreoid gland, and we can only excuse the appearance of yet another volume on the subject by the late Dr. E. P. Sloan[1] because it is put forward as a memorial to his work on this particular gland. Dr. Sloan died while the book was in preparation, and it has been edited by the members of his clinic.

In a foreword it is pointed out that the Sloan Clinic, which was organized in 1917, became one of the American national centres for the treatment of thyreoid disease. It is estimated that up to the end of 1934 over 20,000 cases of goitre were registered, and that over 15,000 operations were performed on this gland at the clinic. In 1923 Sloan founded a group composed of men who were particularly interested in thyreoid disease, and from this small nucleus the American Association for the Study of Goitre was formed in 1924. Later, in 1927, he headed the American delegation to the first international goitre conference, held in Berne, Switzerland.

We agree with the sentiments expressed by Sloan in the preface written by him, while preparing the volume for publication, that a work on thyreoid diseases should discuss goitre and its problems from the viewpoint of all whose efforts are directed towards the elimination or improvement of thyreoid ailments, that the future thyreoid control should be the province of preventive medicine, and that this goal will be reached only through the cooperation of all surgical and medical agencies. He rightly points out that the principles of preventive medicine, properly applied to the goitre problem, can save innumerably more lives than can the surgeon's art.

To recount the working theories and conclusions of a surgeon who directed twenty-five years of his life exclusively to goitre work is said to be the aim of this book, which is not claimed to be encyclopædic. The physician and the public health worker may find cause to quarrel at the author's conclusions, and the surgeon, too, may not agree with his conceptions. The author considered the goitre problem an entity in which there was room enough for all—the surgeon, the internist, the public health official and the psychiatrist—to work together harmoniously for the common good. It is for this reason that he held the opinion that it was well to consider at some length the phases of the goitre question which are often not adequately dealt with in surgical treatises.

The book, with a few exceptions, is an excellent presentation of the present-day aspect towards thyreoid disease, and the illustrations, many of which are coloured, are of first-class order, particularly those in the section on anatomy. Here the author rather specializes in the fascial planes of the neck, which are again emphasized when surgical technique is considered.

In dealing with the pathology we think that the space allotted is not sufficient, and such a condition as thyreoiditis, which, by the way, the author agrees is far commoner than is generally realized, is dealt with in only two paragraphs of ten lines. No mention is made of the lymphatic type of thyreoiditis as described by Hashimoto. The operative technique is described simply but effectively, and the illustrations again are excellent. The author is an advocate of the mid-line split in place

[1] "The Thyroid: Surgery, Syndromes, Treatment", by E. P. Sloan, M.D., with a foreword by W. S. Bainbridge, M.D.; 1936. London: Baillière, Tindall and Cox. Double crown 9mo, pp. 483, with illustrations. Price: 45s. net.

of the cross-cutting of muscles, and he emphasizes the importance of recognizing the fascial planes as a great help in the operation.

We notice that there is no mention made of the operation of complete thyreoidectomy, either in the treatment of certain forms of heart disease or in advanced thyreotoxicosis, and the avoidance of injury to the superior laryngeal nerve in dealing with the upper pole is not emphasized as it should be.

There is an important chapter on the parathyreoid and the thymus glands, and at the end of the book there is an historical chapter of fifteen pages. A chapter is given up to nomenclature and classification, and there is a bibliography of thirteen pages, also an index of authors, index of subjects, and an index of names.

We notice, as is so common among American publications, that very few of the present-day British workers in the subject have received mention; particularly do we miss the name of Sir Thomas Dunhill, who is one of the pioneers of local anæsthesia in operations upon this gland.

The book is an excellent one and we recommend it accordingly. We cannot, however, help calling to mind the words: "Of making many books there is no end, and much study is a weariness to the flesh."

A TEXT-BOOK OF SURGERY.

THE fourth edition of Homan's "Textbook of Surgery" brings up to date this successful work from the Harvard school.[1] The author, as in previous editions, has drawn upon material from twenty-three members of the surgical department of the Harvard Medical School.

He is to be congratulated on the way regional work from so many sources has been adapted and systematized into a comprehensive and easily read volume. Although rewritten and containing an additional chapter on "Amputations and Plastics", the format and size remain much the same as in previous editions. The subject matter is presented in narrative style, and the short historical sketches at the beginning of each chapter, and personal touches throughout the text, make the reading a pleasure. Arresting sentences are scattered throughout; for example: "In surgery, however, as in every other art, fundamental matters are perennially being discovered, discredited, forgotten, discovered and reaffirmed."

That the work is entirely up to date is seen after a perusal of such sections as "The Treatment of Burns", "Megacolon", "The Sympathetic Nervous System", "Anæsthesia" and "Plastics". The symptomatology and differential diagnosis are wisely sacrificed for the more important aspects of anatomy and pathology. Outstanding sections are those on peripheral and cranial nerves, the sympathetic nervous system, first aid to fractures, surgery of abdominal organs, hernia and plastics. The illustrations are a feature of the book and consist of over 500 line drawings, which are well chosen and clear. The student will find this book a great pleasure to read; the sound surgical principles and teachings of eminent contributors are presented by the author in a most palatable form.

THIRTY YEARS OF PUBLIC HEALTH WORK.

SIR ARTHUR NEWSHOLME may look back upon a long life spent at work in the field of public health.[2] The marvellous progress made in these services in England since the beginning of the century will be a lasting monument to his work during his term of office as principal medical officer of the Local Government Board. The guiding principle of Sir Arthur Newsholme's work has been his recognition of the value of prophylaxis. This is plainly evident if we study the memorandum, issued in October, 1918, on the subject of the influenza epidemic of that year. This recommends the continuous ventilation of houses and places of public resort as the most valuable means of suppressing the spread of epidemic catarrh, and stresses the importance of avoiding overcrowding, dirt and haphazard spitting. Again, the author is insistent upon the danger to the community, during such epidemics, of the lack of control over alcoholism and over the misguided persons who disseminate septic conditions of the nasopharynx.

Sir Arthur Newsholme takes the view that one of the chief advances in public health administration in the future will be directed towards the securing of improved environment and hygienic control of the general population. Only by this means will it be possible to prevent the continuous recurrence of illness. This will involve the scientific treatment of root causes and will abolish the empirical treatment of a train of symptoms.

It is mostly due to the unremitting labour of Newsholme that maternal and child welfare services, as well as the control of tuberculosis, have become governmental or municipal activities.

In a general discussion on some aspects of national sickness insurance, the author makes the point that domiciliary medical services are productive of grave delays in treatment and so prevent early recovery from illness. The insurance services of Great Britain are, he considers, less far-reaching and narrower in scope than those which exist in many European countries. They contain scanty provision for the obtaining of consultants' opinions, and few facilities for pathological investigation. This means that patients suffering from any illness of more than the most trivial kind must automatically fall back upon voluntary hospitals, which are commonly overcrowded.

In his concluding chapters, Sir Arthur Newsholme writes interestingly of his official duties in Whitehall, and of visits paid on missions of inquiry to Soviet Russia and America.

Notes on Books, Current Journals and New Appliances.

BRITISH GIANTS IN MEDICINE.

DURING 1934 and 1935 a series of articles appeared in *The Medical Press and Circular* under the title "British Masters in Medicine". These articles, to the number of 24, have been reprinted under the editorship of Sir D'Arcy Power.[1] They "are written by those who have been attached to the great institutions which their heroes made famous". The book has 253 pages; the individual chapters therefore are not long, but generally speaking they set out in an interesting fashion the character, work and worth of the personages discussed. We read of Harvey, Sydenham, Floyer, Cheselden, Pott, Hunter, Lettsom, Jenner, Willan, Bright, Addison, Stokes, Ferguson, Todd, Simpson, Paget, Lister, Turner, Thomas, Robert Jones, Manson, Osler, Mackenzie and Starling.

As Sir D'Arcy Power states in the preface, "the record proves that the flame of genius still burns fiercely amongst us and the praise of these men is an incentive to their successors". This book is easy to read and is attractively set up by the publishers; it will give many a half-hour of pleasure to those who buy it.

[1] "A Textbook of Surgery", compiled by J. Homans, M.D.; Fourth Edition; 1936. Springfield: Charles C. Thomas. Double crown 9mo, pp. 1274, with illustrations by W. C. Shepard. Price: $8.00 net.

[2] "The Last Thirty Years in Public Health: Recollections and Reflections on my Official and Post-Official Life", by A. Newsholme, K.C.B., M.D., F.R.C.P.; 1936. London: George Allen and Unwin Limited. Demy 8vo, pp. 410, with illustrations. Price: 15s. net.

[1] "British Masters of Medicine", edited by Sir D'Arcy Power, K.B.E., F.R.C.S., F.S.A.; 1936. London: The Medical Press and Circular (Baillière, Tindall and Cox). Demy 8vo, pp. 253, with 32 plates. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, APRIL 17, 1937.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

NEWS, NEWSPAPERS AND MEDICAL PRACTITIONERS.

The recent publication in certain newspapers of pictures of patients lying in bed in hospital after sustaining severe injury in accident has again focused attention on the relationship between medical practitioners, their patients and newspaper journalists. There was a time when newspaper editors were satisfied to discover the name, age and address of any person who was hurt in an accident, and to publish all these details with a list of the several injuries sustained. This practice was objectionable enough, but now the newspaper photographer enters a hospital ward and secures a picture of the injured person; the more bandages that are to be seen, the more anxious and harassed the patient's expression, the better. The present pictures might be improved if the patient's wife sat in the background with an agonized look on her face, and his little daughter stood, tearful, in front of the bed, carrying an untidy-looking doll. If the patient had no daughter, the photographer should have little difficulty in finding someone else's child who would, for a consideration, pose for the camera. For many years we have had public hospitals; now, apparently, we have public accidents as well. We cannot say that the present custom speaks well for the journalism of today.

The average man who seeks medical advice is sensible enough to know that he would prejudice his own safety were he to withhold anything that would help in the making of a correct diagnosis or in the planning of efficient treatment. But it probably does not enter his head to make any reservations, for the tradition of medicine is such that most men entrust to their doctor's knowledge not only the details of a present illness, but also any relevant facts from their past lives, and, if need be, their inmost thoughts. It is by this confidence between patient and medical attendant that the successful practice of medicine is made possible. Every medical practitioner instinctively resists the inquiries of a meddler as to the nature of a patient's illness or its causation. Even in a court of law, when directed by a judge to give information that he has received in confidence from his patient or that he has discovered in the course of his clinical examination, he will obey the judicial order under protest. If the patient gives permission to the medical attendant to impart certain information, it is quite another matter; but even in these circumstances the medical practitioner will be wise if he has permission stated in writing. The obligation on a medical practitioner to respect the confidence of his patient rests equally on hospital authorities and on nurses and others whom they may employ. When an accident occurs and patients are admitted to hospital, details of the patient's injuries and of his condition are given to the newspapers. This information is sometimes obtained from attendants at the hospital, medical or otherwise; it may be obtained from sources outside the hospital. If the information is given by the medical attendant or anyone else connected with the institution, the patient's confidence has been betrayed. If the medical attendant gives the information, he breaks his Hippocratic oath "that all such things should be kept secret"; if he acquiesces in the betrayal of confidence by someone else, he is equally culpable.

But, it may be objected, many patients have no dislike of seeing either their names or their pictures in a public newspaper. Apart from the fact that there are many patients who do object to this kind of thing, the argument is not valid. To betray a confidence without permission, even though the person betrayed does not object, is none the less a betrayal.

This matter is of no small importance; it strikes at one of the foundations of medical practice. It is one that should be taken up by Branch councils or their hospital subcommittees. The medical superintendents of the large metropolitan hospitals should be approached in the first instance; the honorary staffs should also be asked to consider the question. A regulation should be introduced and enforced, forbidding the giving of any information about a patient's injuries to those not entitled to receive it. The prohibition would naturally cover the inquisitive adventures of newspaper photographers. The resident medical officers should have their attention constantly drawn to the seriousness of any departure from the high standard set out in the Hippocratic oath. If a definite stand were taken by metropolitan hospitals, other institutions would soon fall into line. To discuss this kind of thing with newspaper editors is quite useless.

Current Comment.

FATIGUE IN CHILDREN.

G. A. Lamont[1] has recently presented a paper on fatigue, based on the study of 125,000 children. The term "fatigue", be it noted, denotes a state induced in nervous and muscular tissues by changes resulting from activity. "Tiredness" is the name given to that sensation which, though commonly produced by fatigue, seems to exist sometimes quite apart from any activity-produced fatigue.

The changes of fatigue are due to exhaustion of those substances in the muscle which are needed for the supply of energy, or, more exactly, to an accumulation of sarcolactic acid in excess, of which the body has not ridded itself. If the amount of the acid which is produced can be removed by the processes of oxidation, the phenomena of fatigue are observed only when other factors causing fatigue, such as lack of bodily fuel, insufficient loss of heat and moisure, or monotony, have to be considered.

[1] *Canadian Medical Association Journal*, January, 1937.

The clinical picture of fatigue is especially noticeable during the course of serious illnesses, such as the anæmias, diabetes and tuberculosis, in which the smallest nervous or muscular activity may lead to its appearance. In these states of morbid fatigue, infections of the respiratory tract seem to play a leading part, especially when chronic diseases of the accessory sinuses have been a long-standing trouble. Digestive disorders, associated with excessive carbohydrate intake and a deficient protein supply, are also important causal factors. Defective posture, with a consequent upset of the bodily mechanics, is hardly of less importance.

Juvenile obesity, as a cause of fatigue in both infancy and youth, comes commonly under the notice of the pædiatrician, and certain degrees of the disorder are properly considered under the heading of endocrinology. Nevertheless, we have to recognize the existence of a form of obesity in childhood which results from gross over-eating, as well as the endocrine varieties of the disease, which are due chiefly to involvement of the thyreoid and pituitary glands. In both girls and boys, from one or other of these two causes, there may result a general adiposity involving the carrying of extra weight out of all proportion to the age group and the general size. Children thus afflicted easily become fatigued, the more so as they usually attempt to compete with children who are their seniors in point of years. Small, under-developed children also present the phenomena of fatigue not uncommonly. In these children the cause may be found in the endocrine chain, as in cases of infantilism. But nutritional factors, due to a lack of lipoids, are also to be discovered. This want of lipoids may be the result of the common infectious diseases of childhood, or of tuberculous or syphilitic infections.

But many children, free from gross pathological taint, are habitually fatigued. In small children of pre-school age the trouble may be caused by lack of the necessary afternoon rest; in older children late hours are chiefly responsible. The healthy child should be his own pace-maker, but the conditions of modern life are such that the life of a child of today is too full; his programme is mapped out from hour to hour, whether he is at work or at play; he meets too many people; he is prevented from adopting the method of living, natural to the child, which turns the day into a series of alternating periods of activity and self-imposed idleness. In children, too, hunger is a powerful, though frequently unrecognized, cause of fatigue. This form of fatigue may arise not from any deficiency in the daily supply of calories, but from a lack in the caloric intake even from hour to hour throughout the day. In young school children this form of fatigue is especially to be observed. The child who takes a poor and hurried breakfast and an insufficient luncheon affords a typical example, as does the youngish girl who is "slimming", and the athletic boy who is anxious to keep down his weight.

During the taking of strenuous exercise, lactic acid is so rapidly produced that it cannot be eliminated. In "sprinting" athletes, it has been estimated, one gramme of the acid accumulates at every stride, an amount equal to 40 grammes in a dash of 100 yards. These biochemical changes are productive of intense fatigue. In long-distance runners the hunger factor becomes part of the clinical picture, and an interesting correlation occurs between the phenomena of hypoglycæmia and fatigue. The result of proper athletic training seems to be to accustom the circulation to the removal of sarcolactic acid and even to train the muscles to tolerate this product of fatigue.

It may be said that much of the fatigue of childhood might be prevented by applying to school life many of the principles which obtain in industry, such as the control of lighting, of air flow, of temperature and moisture, of noise, of posture while at work. We are unable to say, so far as children are concerned, where natural fatigue ends and abnormal fatigue begins, and we have still to determine how much the mental element enters into the two conditions.

CORONARY OCCLUSION IN YOUNG PEOPLE.

It is well known that cardiac infarction may occur in very young persons, though it is regarded as an unusual event. In museums hearts of young children are occasionally seen showing a fatal infarction of the myocardium. But it is of importance to have some idea whether the occurrence of such an accident is so excessively rare even in young adults as to be negligible from the standpoint of diagnosis. Levine and Brown, in their classic account of coronary occlusion published eight years ago, recorded only three cases occurring in persons under forty years of age out of a total of 145, and found none in persons under the age of thirty-five. T. M. Durant, in writing on this subject, quotes other similar reports from the literature.[1] His own series of case histories presents a different aspect of this subject, for he found that seven out of 114 proved cases of occlusion in the University Hospital Ann Arbor were observed in patients under the age of thirty-five years. The ages of these patients were 23, 33, 31, 35, 33, 31 and 33. The histories are all more or less characteristic, and adequate electrocardiographic confirmation of the diagnosis was obtained. Two patients died, and autopsy revealed a large infarction of the heart muscle in each case. There are two interesting points that call for further attention: the question of diagnosis and the elucidation of the cause. Diagnosis really does not present any greater difficulty than in older patients; the only difficulty lies in the mental barrier of scepticism in the mind of the physician. Possibly ten years ago we could associate cardiac infarction only with known and gross vascular disease, and we refused to entertain such a diagnosis in a young and presumably healthy person. Now we realize that coronary occlusion may sometimes occur without the manifest gross arterial disease, and it may be arteriolar rather than arterial. The second point concerns the underlying cause. In three of the seven cases no cause could be found; there was no evidence of diabetes or of hypertension or other vascular disease, nor was there a significant familial history. In the other cases two patients were of the hypertensive group, one was a mild diabetic and the other suffered from syphilitic arterial disease; this last is interesting, as syphilis is a known but not usual cause. All the patients were males, and none was obese; the majority were engaged in laborious work; in no case was there a history of a previous infection.

Here it is interesting to refer to some work published recently on hæmorrhagic reactions found in the intima of atheromatous coronary arteries. J. C. Paterson has studied a series of hearts the subject of infarction, and found that in these cases vascularization of the intima with capillary channels had taken place and small hæmorrhages had occurred from these tiny vessels.[1] This was thought to be the actual source of the thrombus in these cases. Paterson found these lesions only in atheromatous arteries, and especially in those the subject of thrombosis. They did not appear in normal vessels. He considers the sequence of events to be endarteritis with formation of capillary channels in the intima, atheroma and softening, and capillary rupture. It is possible, then, that this may be the mechanism by which quite a small lesion in a coronary artery may initiate a thrombosis.

But, quite apart from pathological discussions, the possibility of coronary accidents in the young subject should not be overlooked. It is sometimes said of a patient: "But he is too young to have a coronary occlusion." This is not necessarily true, and though such an event is probably unlikely to occur in a young and apparently healthy person, it is by no means impossible.

[1] *Annals of Internal Medicine*, January, 1937.

ADELAIDE AND THE CONGRESS.

Attention is drawn to the article on Adelaide, its history and surroundings, but Dr. A. Grenfell Price, that appears in this issue. We feel confident that this contribution will be a stimulus to many who may be hesitant about enrolling their names as members of congress. We are grateful to the South Australian Publicity and Tourist Bureau and to the University of Adelaide for supplying the photographs reproduced in this issue. Further, on our behalf and on that of the Executive Committee of Congress, we wish to express our appreciation of the generosity of Commonwealth Serum Laboratories in donating the space on the front cover so that we might reproduce one of the Adelaide pictures.

[1] *Archives of Pathology*, September, 1936.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Ulcerative Hodgkin's Disease of the Skin.

F. E. Senear and M. R. Caro (*Archives of Dermatology and Syphilology*, January, 1937) describe a case of Hodgkin's disease in which the condition manifested itself as ulceration of the skin. They also state that the ulcerative type of cutaneous lesion in Hodgkin's disease is not mentioned in all dermatological text-books. The ulceration may occur in three different ways, the processes being as follow: (*a*) ulceration of limited extent developing in a number of small nodules in the skin; (*b*) extensive ulceration developing in the skin by extension from underlying lymph glands, bones and other tissues; (*c*) extensive ulceration developing in large infiltrations appearing in the skin without involvement of the underlying structures. Ulcers may be single or multiple and may vary in size and shape. They bleed freely when touched, and as a rule are painful. The submaxillary, clavicular, axillary and thoracic regions are most commonly affected, although no part of the body is exempt. These ulcers may be difficult to distinguish from the lesions of syphilis, sarcoma, *mycosis fungoides*, epithelioma and tuberculosis. The ulcerative lesions usually develop after the glandular involvement has become apparent, but in some cases they have furnished the first evidence of the disease. The histological picture of tissue removed from the ulcer is usually characteristic of Hodgkin's disease, the number of Reed-Sternberg cells varying greatly in different cases. In some instances, however, no typical changes are apparent and the picture may be strongly suggestive of sarcoma.

Poikiloderma-Like Dermatosis.

F. E. Cormia (*The British Journal of Dermatology and Syphilis*, January, 1937), writing on the subject of poikiloderma-like dermatoses, reports a case with unusual localization and atypical features. The patient was a medical practitioner, aged thirty-five years. The eruption covered the entire surface of the palms of the hands and the palmar surfaces of the fingers and thumbs. The main pathological changes were multiple, small, irregular areas of vascular dilatation. Only one definitely purpuric spot was seen. Had it not been for the vascular changes, the appearance would have simulated scleroderma, the skin between the vascular lesions having a dull whitish look. The entire area felt hard. Superimposed on the atrophic-looking substratum was a considerable degree of scaling. The eruption was rather sharply demarcated. There was also present on the trunk and proximal part of the extremities an asymptomatic follicular eruption. Each follicular lesion consisted of a hyperplasia around the individual hair follicles. There was an appearance of central atrophy in each follicle. These lesions were similar to those described by Jacobi in his original case, and, the author thinks, may correspond to what Gougerot and his co-workers believe to be the initial lesion of poikiloderma. The author gives a full description of the microscopic appearances. He summarizes them by stating that the disease was of an atrophic and telangiectatic nature, which could be reconciled with poikiloderma. The hardening, which had the clinical appearances of scleroderma, did not appear to have been produced in the same way as that of scleroderma—it was not the sequel to an inflammatory process. The author points out that this case is an example of the vague and ill-defined character shown by many of the poikiloderma-like dermatoses. He classes it as a variant of the original Jacobi syndrome and clinically similar to the scleropoikiloderma of Richter.

Nævocarcinoma of the Skin.

I. G. Williams and L. C. Martin (*The Lancet*, January 16, 1937), writing from the Middlesex Hospital, discuss nævocarcinoma of the skin and mucous membranes. They base their remarks on 25 cases. Of the 25 cases, 20% arose as a result of malignant change in a previously benign melanoma. The authors describe the histological findings and the results of treatment; and they suggest: (*a*) that the non-pigmented papillary "squamous" cell growth is the most malignant, (*b*) that the pigmented masses of "spheroidal" cell growth are the least malignant. They add that the presence of pigment in a growth appears, other things being equal, to be more favourable than its absence.

Shoes and Ringworm of the Feet.

R. C. Jamieson and A. McCrea (*Archives of Dermatology and Syphilology*, February, 1937) have undertaken an investigation of the possibility that shoes act as a residual source of infection in ringworm of the feet. They describe their present communication as a preliminary report. In order to determine how many patients with ringworm harboured parasitic fungi in their shoes, scrapings were obtained from the inner soles of shoes of patients who had a definite fungus infection of the feet, proved clinically and in most instances microscopically. Fifty-three samples of scrapings were examined by cultural methods. Sixteen samples yielded pathogenic fungi in culture; thirteen samples yielded no fungi; culture of twenty-three samples gave doubtful results, and culture of one control sample gave negative results. Known pathogens were isolated in sixteen instances; twenty-eight specimens showed suspected pathogens, and various fungi believed to be non-pathogenic were found in large numbers, many specimens revealing a number of varieties. Although definite pathogens were found in sixteen specimens, and suspected pathogens in twenty-eight, the authors believe that infected shoes can be considered only as potential sources of infection or reinfection in ringworm of the feet. Reports from patients who were studied revealed that irregular recurrences of moderate severity had been the rule, regardless of whether new or old shoes had been worn. The authors find it impossible to state definitely whether fungi found in scrapings from the inner soles of shoes had been deposited there from the infected skin or had been carried there from the stockings.

UROLOGY.

The "Cord Bladder".

By the use of his tidal drainage apparatus, Donald Munro (*Journal of Urology*, December, 1936) claims to have reduced the incidence of urinary sepsis in "cord bladders" from 73% to 15%. For purposes of prognosis and treatment such bladders are classified by cystometric examination into atonic, autonomous, hypertonic and normal. All types are amenable to treatment by tidal drainage provided the apparatus is adjusted to suit the individual type under treatment. The end results of a "cord bladder" due to spinal injury are: uninhibited "cord bladder" in cases of transection above the sacral segments; autonomous "cord bladder" in cases of destructive lesions of the sacral segments or *cauda equina*; normal "cord bladder" in all other spinal cord injuries. In addition to the great reduction in the incidence of infection, the problems of nursing in cases of spinal cord injury are simplified and the occurrence of decubitus ulceration is rare.

Total Cystectomy.

Bernard Ward (*Proceedings of the Royal Society of Medicine*, December, 1936) records seven successful cases of total cystectomy for vesical tumour. He points out that the gravity of the operation is much less when it is performed in children for congenital malformations than when it is done in adults—usually elderly—for carcinoma. His indications are: (i) infiltrating growths of the bladder neck or base; (ii) extensive infiltrating growths of the lateral and posterior walls which cannot be excised locally with sufficient healthy tissue; (iii) extensive multiple sessile papillomata; (iv) large multiple infiltrating papillary carcinomata; (v) rapidly spreading carcinoma of the mucosa and submucosa—a rare type; (vi) some cases of extensive leukoplakia and malakoplakia, which are undergoing malig-

nant degeneration; (vii) malignant growths of the prostate, confined to the superior surface and neck of the bladder. The author considers that, for these cases, diathermy, radium, radon and deep X ray therapy have failed. But before the major proceeding is undertaken, other factors must be considered: (*a*) the age and general build of the patient; (*b*) the heart and lungs, which must be healthy enough to carry him through a severe surgical procedure; (*c*) adiposity, which is a contraindication; (*d*) the state of the kidneys and ureters. The author prefers to implant both ureters at one sitting into the large bowel, the right being anastomosed first. The second stage consists in removal of the bladder. For the implantation he uses the Coffey technique, with the variation that a small rubber catheter is used as an internal splint during the implantation of the ureter and subsequently manipulated into the rectum. No catheter is left in the ureter. In performing cystectomy, Ward prefers the transperitoneal operation performed through a transverse incision with partial division of the recti muscles.

Uretero-Ureteral Anastomosis.

C. H. NEUSWANGER (*New England Journal of Medicine*, October 8, 1936), after experiments on thirty-two dogs, concludes that the simplest and most efficient method of uretero-ureteral anastomosis is performed as follows: The diseased ureter is cut obliquely; a silk suture is placed in its tip, and, after incision of the recipient ureter, the suture is passed through its wall from within outwards, drawing the divided ureter into the lumen of the recipient ureter. The suture is tied on the surface of the recipient ureter. A successful case is reported in the human subject.

Pyeloplasty.

J. K. ORMOND (*Journal of Urology*, November, 1936) gives an account of six unsuccessful operations for non-calculous hydronephrosis, representing one-third of his cases. The study of these cases and those recorded in the literature lead him to the following conclusions. (i) He distrusts the Heinecke-Mikulicz type of operation, believing it to be suitable only for early uninfected cases, for which reimplantation of the ureter has frequently given excellent results. (ii) Uretero-pyeloplasty has no advantage over reimplantation of the ureter. It is difficult to perform with precision, owing to the displacement of the kidney for the exposure; the line of incision and repair may be distorted when the organs are replaced in their normal positions. (iii) For ureteropelvic junction obstructions, resection of the junction is favoured. The ureter, cut obliquely, is reimplanted in the most dependent position. (iv) For reimplantation the author's technique includes accurate apposition of the cut edges without any protrusion of the ureter into the pelvis. A splinting ureteric catheter is always used and remains in position about three weeks. (v) In the presence of acute or marked infection, preliminary nephrostomy is to be performed. (vi) Nephropexy is performed in every case in which the kidney is not bound down by adhesions. (vii) Plastic operations are to be reserved for those patients in whom conservation of the renal function is imperative, or for those in whom the desirability of conservation of the renal function outweighs the chance of increased expense, danger and loss of time due to an unsuccessful operation.

Prostatic Resection.

M. STERN (*Urologic and Cutaneous Review*, November, 1936), who introduced the resectoscope ten years ago, reviews its present status. He deprecates the published statements of those who advocate resection for all forms of prostatism, and of those who deny the necessity for preliminary preparation. In his view, resection is undoubtedly indicated in the bulk of all prostatic cases, but it must be conceded that it has been found inadequate in some cases and too radical in others. Enucleation is reserved for cases of "advanced prostatism", the chief indications being an extensive tumour or one of the forms of gross sepsis. Carcinoma, bars and contractures of the bladder neck and the majority of adenomata are suitable for resection. For the early cases with little tumour formation the author advises his prostatic resorption technique.

Differential Diagnosis in Diseases of the Testis and Epididymis.

M. BONINO (*Zeitschrift für Urologie*, May, 1936) states that acute, subacute and chronic infective changes in the organs contained in the scrotum are frequently, but mostly erroneously, labelled orchitis. Most of these infective processes are usually limited to the epididymis. In certain cases the nature of the infective process is very doubtful, and it will often happen that the surgeon desires to be sure whether the process is tuberculous or not. The author proposes a method of "sperm culture" in which the semen is inoculated on media after special preparation and centrifuging. The diagnosis was cleared up by this method in 60% of the author's doubtful cases.

Biological Compensation in Ureteric Obstruction.

N. SERRALLACH, F. SERRALLACH-JULIA AND A. AMELL Y SANS (*Journal d'Urologie*, July and August, 1936) have conducted a series of experiments on guinea-pigs in order to study the measure of defence or compensation occurring in the body after complete obstruction of one ureter. Soon after the obstruction occurs, the intrapelvic pressure rises to a certain degree and the kidney stops secreting urine. The term of cessation of secretion is fixed by circumstances which are not understood, but which are probably of nervous origin. The pressure at which secretion ceases is about 60 millimetres of mercury. A certain amount of absorption of the contained fluid occurs after a while, causing lowering of the pressure, upon which the kidney commences to secrete urine again. A balance between secretion and reabsorption is set up. The chemical composition of the contained fluid depends on the functional state of the opposite kidney, the composition of the blood and the general metabolic activity of the organism. Survival of a completely obstructed kidney is dependent on, first, the advent of anuria in that organ, then on oliguria as regards that organ, later on pyelo-ureteral reabsorption, and finally on the establishment of a collateral circulation. Generally speaking, pain does not follow sudden complete obstruction of the ureter by a ligature, for reasons given above. Certain spasmodic pains may occur, however, since the ureter, being a muscular canal, is subject, like other muscles, to spasmodic cramps.

Vesical Neoplasms.

J. M. IRGER AND A. I. MICHELSON (*Journal d'Urologie*, June, 1936) consider that biopsy does not always settle the question of the benignity of vesical papillomata, for the malignant portions may not be included in the portions removed for examination. In any case, papillomata which are histologically benign throughout cannot always be considered benign clinically; indeed, their tendency to recurrence after open operation makes it necessary that they should be considered papillomata of the bladder, in the category of malignant tumours. The best method of treatment of papillomata is cystoscopic electrocoagulation, and when this is not practicable, transvesical electrocoagulation should be performed. So far as definitely malignant tumours are concerned, partial resection of the bladder is applicable if the tumour is on the anterior or lateral walls of the bladder. In all other cases the only operative treatment of any use is total cystectomy, after implantation of the ureters. In place of cystectomy palliative measures may be used, namely, electro-coagulation and radium therapy.

Pathogenesis of Prostatic Hypertrophy.

C. SPANGARO (*Urologia*, September, 1936) passes in review the various theories of the pathogenesis of prostatic hypertrophy and declares that the theory of neoplastic myomatous formation is the most accredited among recent authors. From his personal researches the author favours the theory of a prostatic myomatosis of multicentric periurethral origin.

Special Article.

SOUTH AUSTRALIA AND ITS CAPITAL.

The following article on South Australia and its capital, Adelaide, has been written at the request of the Executive Committee of the fifth session of the Australasian Medical Congress (British Medical Association) by A. Grenfell Price, C.M.G., D.Litt., Master of Saint Mark's College, University of Adelaide.

The history of the State and city which welcome the fifth session of the Australasian Medical Congress (British Medical Association) as part of their centenary celebrations, forms an interesting example of man's scientific conquest of a new and strange environment. The foundation of South Australia was the direct result of that scientific progress which produced the industrial revolution with consequent over-population and suffering in the British Isles. The agency was a so-called plan of scientific colonization, evolved by Edward Gibbon Wakefield, to remedy the over-population at home by removing "young marriageable persons" to empty wildernesses overseas.

Wakefield, who was notorious for his abductions and celebrated for his brilliant writings, possessed the gift of attracting and inspiring other brilliant men. From 1830 onwards he gathered around him an able group of young theoretical colonizers, radical in politics and religious outlook, who struggled with the anti-colonizers of the British Parliament and Colonial Office until, in 1834, an Act for the establishment of the "New British Province of South Australia" gave them a field in which to put their ideals to the test. The sale of South Australian land, the application of the proceeds to the immigration of young marriageable persons and the promise of self-government when the Province contained 50,000 persons permitted the trial of principles which were to revolutionize the practice of colonization, not merely in Australia and the British Empire, but throughout the whole world.

Wakefield dominated the foundation of South Australia; Colonel William Light the colonization of a wilderness, unknown save for the brief visits of a few explorers, such as Matthew Flinders, Nicholas Baudin, Collet Barker and Charles Sturt. Soldier, sailor, surveyor, artist and musician, Light, a cosmopolitan genius of European reputation, carried the all-important but crushing burden of selecting the site of the first settlement. Today we know that, in an era prior to the steamship, scientific harbour engineering or modern water conservation, the Adelaide plains, with their fertile soil, safe harbour and girdle of rain-giving mountains, formed the only region which offered any great hopes of success. First, and, at the outset, alone in perceiving the advantages of the area, Light founded Adelaide and maintained his choice against the most bitter and influential opposition. Ruined and hounded to the grave by disloyal employers, and by ignorant opponents, the Surveyor-General maintained his selection and became "the saviour, the preserver of the State".

King William Street, Adelaide.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

The city of Adelaide is Light's memorial. The general scheme may have been formulated in England, but to Light is due the fine location and the magnificent plan

of a double city—North and South Adelaide—separated, and surrounded by 1,700 acres of park lands. It is true that Light made mistakes, such as designing so few north and south thoroughfares, and laying out narrow streets in what is now the main shopping area, where population quickly settled, owing to proximity to the water of the Torrens and the outlets of the coastal roads. But the main faults were due not only to Light, but to later generations who crowded every imaginable public utility into the magnificent space which the Surveyor-General left between the two parts of his double city, and who showed a lamentable absence of ability and foresight in planning many of the suburban areas.

At the outset South Australia encountered grave difficulties. Quarrelsome and incompetent officials, lack of financial resources and niggardly policies nearly wrecked the Province before the settlers evolved pastoral and agricultural methods which suited the Mediterranean environment, and before they discovered the wealth of the Kapunda, Burra and other copper mines. Nevertheless, in spite of the failure of the Wakefield scheme as a mathematical system of colonization, and an almost disastrous financial crisis, South Australia was on its feet by 1844, and by 1851 was a flourishing colony of 66,000 people, planted at a total cost to Britain of £225,000. The scientific outlook of the community was already strongly in evidence. John Ridley had invented the stripper, and Governor Grey and J. H. Dutton had taken steps to promote the artificial fertilization of the soil.

At the close of one hundred years South Australians may recall with gratitude the character of their founders, and examine the effects which the early difficulties had upon the pioneers and the generations which followed them. South Australia is a hard country. Its fertile areas border vast deserts. It is afflicted by periodical droughts and depressions. It suffers from the disabilities of an ill-balanced Federal system, which, in the words of Professor W. K. Hancock, have made the State "a lady of reduced circumstances", and have driven her from the second class to the steerage of the good ship "Commonwealth". Yet, throughout the foundation period—the time of greatest difficulty—the behaviour of this colony of free emigrants was almost exemplary. The Governor, Sir George Grey, a shrewd and cold observer, who saw the pioneers in their hours of deepest suffering and anxiety, summed up their character in striking words: "The calibre of the early settlers gave me trust in the new Anglo-Saxondom of the Southern Hemisphere. There was a worth, a sincerity, a true ring about them which could not fail of great things."

Victoria Square, Adelaide.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

The middle period of South Australian history (1850-1880) was marked by the bestowal of self-government (1856) and by two remarkable efforts to expand. As early as 1840 the South Australian Commissioners suggested that, as South Australia had limited resources, the colony should be granted control of what is now Victoria, and in the fifties and sixties it was seen that the flocks and herds were outgrowing the comparatively small area of well-watered land. Hence the colony established and developed steamship trade on the Murray during the fifties, and acquired the Northern Territory in the following decade. Both efforts failed, the first through geographical conditions and the growth of railways fostered by the jealousy of other colonies; the second because the

Cherry plum blossom, Mount Lofty Ranges.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

Northern Territory is a land of poor soil and uncertain rainfall, where, at far greater expenditure than that incurred by South Australia, the Federal Government has met with singularly little success. Federation, achieved in part by the efforts of South Australians, helped to redeem both failures. The Murray Waters Agreement of 1915 promoted irrigation and added a fertile province to the State. In 1911 an unwilling nation took over the burden of the Northern Territory.

From 1880 to 1910 South Australia passed through a period when it seemed that the colony had reached its maximum of prosperity and might even decline. The people, however, called upon science to remedy their dwindling stock and exhausted soils, and the colony entered upon a new and unexpected advance. Two aspects of this progress are particularly interesting. The first was the growth of agriculture and of farming population, due to increase of the wheat industry, which supplanted

pasturing in the moister areas and extended into the drier lands with the growth of railways, of scientific farming and of machinery. The second was an advance beyond the primary producing stage to that of secondary industry, under which the population of Adelaide increased with great rapidity and after 1917-1918 exceeded that of the country districts.

Invigorated by prosperity, the State faced the Great War and the world depression as a strong unit, in a strong nation of the British Commonwealth. In the War, 29,000 men or 8% of the population, sailed for the various fronts, 5,352 made the supreme sacrifice, and in a list of 1,600 decorations five were the Victoria Cross.

A century of South Australian history shows on the whole the satisfactory adaptation of a northern European people to a Mediterranean environment. It seems fair to claim that later generations of South Australians have maintained the pioneer heritage. In every aspect of national life—in the creation of a united Australian Nation, in the sacrifices of the Great War and great depression, in social legislation and betterment, in education, in philanthropy, in scientific progress and invention, in aviation, in sport—the descendants of the pioneers have kept faith. It is true that some scientific observers, such as Ellsworth Huntington, consider that the hot summer may prove deleterious, but this view probably under-estimates the influence of the cool winters and of the frequent changes of weather, brought by the anti-trade winds. Statistics are notoriously misleading, but it may be noted that the figures for death rates, serious crimes, drunkenness, illegitimacy, insanity and suicide are less than the Australian averages, and in some cases are the smallest for any Australian State. At present the natural increase and the birth rates are lower than the Australian average, and it is a serious matter that in a region which was marked in its early years by a high percentage of young people, the percentage of children in the population should show a steady decline. The substitution of bath-chairs for perambulators is, however, characteristic of Western civilization, and the natural increase in South Australia is higher than that of most of the countries of Europe.

King William Street, Adelaide, from the Town Hall balcony.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

Dr. F. S. Hone points out in the "Centenary History of South Australia" that, when the colony was founded, the land was free from epidemic or endemic disease, and that the fine climate, the youthful, exotic population, and the small number of aborigines were all conducive to good health. Before long, however, infections were introduced, but cholera and typhus never gained entrance; bubonic plague affected Port Adelaide only once, and for a brief season, and smallpox secured only a temporary and local footing. Tuberculosis appeared at the beginning of the colonization and became serious in the latter half of the nineteenth century, owing to the indiscriminate dispatch of consumptives from Britain. Diphtheria, scarlet fever, measles and whooping cough appear at intervals; but gastro-intestinal infections have been the worst scourges, typhoid fever being particularly serious until recent times, when the better control of water and of food supplies, together with immunization methods, practically eliminated the disease.

From the earliest days South Australia has had the advantage of efficient medical scientists, who have played, and still play, a leading part in fostering community interests. The growth of hospitals dates from 1840; water sewerage in Adelaide from the Act of 1878; and from 1885 the medical school of the University of Adelaide has conducted efficient professional training in medicine and steadily developed work in medical research. Under the guidance of the medical scientist the crude death rate has fallen during the last fifty years from 15 deaths per thousand of population to 8·5 in the last quinquennium. Under the same scientific guidance infant mortality has been reduced, until in 1933 it was as low as 33 per thousand, a figure which only New Zealand seems to equal in the world.

For the next century South Australia can anticipate further progress, although her population will face two serious difficulties: the expected fall in European population, with the consequent contraction of markets, and the persistent, but not unnatural, drag of secondary industries to the eastern States. In spite of these problems, it seems probable that the State will continue to advance, for only some 50% of the agricultural lands are as yet cultivated, large tracts of irrigable country will be opened up on the River Murray, and scientific manufacture should increase the population of Adelaide to a considerable extent. While South Australians must confess that natural resources are more limited than those of any other mainland State, the close of the second century may see an increase of at least 100% in the population of Adelaide and of the country

districts, and a total population of over 1,000,000 people in the State.

Owing to its command of the comparatively small area of well-watered country in South Australia, the Adelaide metropolitan area now contains about 318,000 people, some 54% of the population of the State. The northern and southern parts of the city, as laid out by Colonel Light, contain 1,042 acres, and are surrounded by park lands, which also separate North and South Adelaide, and in all contain 1,710 acres. The parks, which divide the two sections of the city, are the site of many of the chief public utilities. The Railway Station, Government House, the Public Library, Art Gallery and Museum, the University, the School of Mines, the Adelaide Hospital, the Botanic Gardens, the Adelaide Oval, the Zoological Gardens and the Municipal Golf Links are picturesquely situated on the slopes between North and South Adelaide or on the flats bordering the artificial lake which has been constructed on the River Torrens. Other public utilities, such as the Victoria Park Racecourse, the West Terrace Cemetery, the Observatory, and numerous sports grounds, are located in the encircling girdle of parks. The people of Adelaide prize their parks greatly. They regard them as the "lungs of the city", and strenuously resist any encroachment. The Torrens Lake, created by the building of a weir, and opened in 1881, has recently been greatly improved by the City Council, which has laid out several beautiful gardens along its banks. Winter floods and the silt problem, unfortunately, compel the opening of the weir, and for some months each winter reduce the waterway to a shallow stream flowing through ugly mud banks. Nevertheless, the lake is charming in summer and is very useful for sport, as it provides a course of about a thousand yards for racing eights. The Adelaide Oval, near the northern bank of the Torrens, is generally conceded to be one of the most beautiful cricket grounds in the world, with its fine vista of hills in the distance. Near the oval, the Anglican Cathedral, with its twin spires, occupies a fine site, commanding King William Street, the link between the two parts of the city.

Colonel Light laid out Adelaide in rectangular form, with the streets cutting at right angles. South Adelaide is the principal business area, North Adelaide being chiefly residential. The main shopping area lies in South Adelaide, and developed early, owing to the water control of the Torrens. Some of the finest residences are in North Adelaide, on the edge of the scarps which look south and east towards the mountains. In accordance with contemporary ideas of town planning, Colonel William Light gave Adelaide streets of varying width, and this has created serious traffic problems, as the main shopping area grew up on the narrow streets close to the river. The first Surveyor-General caused further traffic difficulties by laying out only three main streets in a north-south direction, it is believed because he was anxious to check the effects of the hot north winds. In spite of these faults, the plan of Adelaide, with its squares, its wide central streets and its parks and gardens, has led to the evolution of a very beautiful city.

The city of Adelaide now contains some fine buildings. Visitors should note the Railway Station, Parliament House (now being completed, largely through a munificent gift of £100,000 by the Honourable Sir Langdon Bonython), the Bonython Hall and Barr Smith Library of the

North Terrace, Adelaide.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

University, and a number of commercial buildings. The Police Court in Victoria Square, and the old Police Barracks, behind the Museum, are particularly attractive, and the School House of Saint Peter's College, Hackney, built about 1852, is one of the finest examples of college architecture in the Commonwealth.

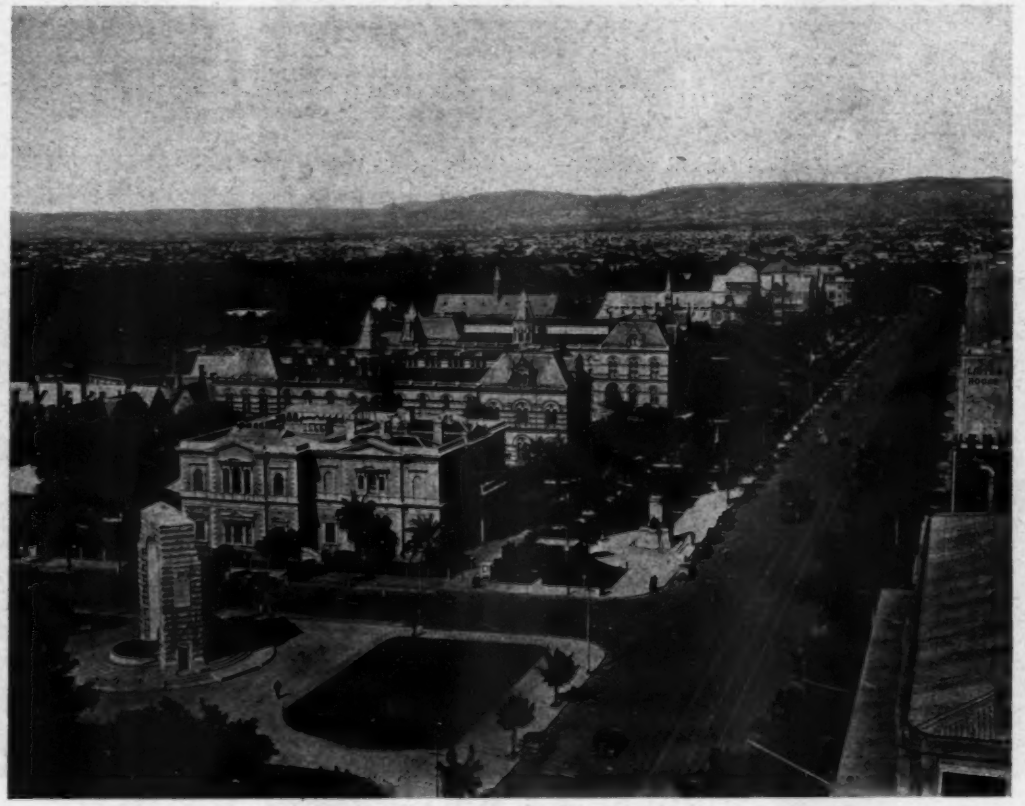
In recent years the University has made remarkable progress under the guidance of Sir George Murray and Sir William Mitchell, as Chancellor and Vice-Chancellor. The institution now possesses a revenue of £115,000 a year, and includes twenty professorial chairs and over 2,300 post-graduate, undergraduate, non-graduating and conservatorium students.

Some years ago the Government granted the University considerable additions of land, and a number of fine buildings have been erected. The southern buildings are of stone. The northern reflect contemporary practice under economic pressure, and are of Georgian architecture in brick. Despite the comparatively small population and slight resources of South Australia, the University contains one of the four medical schools in the Commonwealth. Founded in 1885, The Adelaide Medical School has gained a reputation for the ability of its teachers and the standard of its work. The school now includes three medical professors, four professors of science, who teach junior medical students, more than thirty lecturers and tutors, and about one hundred and fifty students. The work of the three hospital years is mainly conducted by an honorary staff.

A highly important section of the University is the Waite Agricultural Research Institute at Glen Osmond, where some of the most important research work in Australia is being carried out by the staff of the institute and members of the staff of the Australian Council for Scientific and Industrial Research. The institute was founded for the University in 1914 by the generosity of the late Mr. Peter Waite, who made donations to a value of £100,000 for this purpose. The Commonwealth Animal Nutrition Laboratories stand on University property. The system of University residential colleges is less advanced in Adelaide than in Sydney, Melbourne or Brisbane, but Saint Mark's College, situated near the University, on the northern edge of the central parks, contains some sixty resident tutors and students.

Some eight miles from the capital, Port Adelaide, a municipality of some 30,000 people, is located upon the estuary of the River Torrens, although the river itself disperses in reed beds and swamps. The port has followed the usual evolution of river harbours. In 1837 Light established the first landing place at Port Misery, which was situated a short distance above the present harbour, on the first firm ground above the tidal mangrove swamps. As early as the forties this harbour proved shallow and inadequate, and Governor Gawler established the present port, with the help of the South Australian Company, which constructed at great expense a road across the swamps and built a wharf and storehouse. In a very few years it was apparent that even this harbour was situated too far up the river. Frequent redredging failed to keep the channel abreast of the growth of shipping, and the Outer Harbour, where the mail steamers now berth, was constructed at the mouth of the estuary.

The citizens of Adelaide are particularly fortunate in their surroundings. The city is situated some six miles from Saint Vincent Gulf, which is lined for miles by most attractive beaches. These provide such safe bathing



North Terrace, Adelaide.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

that there is only one sea bath, that at Henley Beach. During the hot summer months thousands of people throng the sea front after the day's work. The sandhill country of the coast is ideal for golf. Here are situated the two chief golf courses, Royal Adelaide and Kooyonga, while, close to Adelaide, Mount Osmond and Mount Lofty provide attractive mountain links.

Adelaide is the most fortunate of the Australian mainland capitals in its proximity to mountains. Mount Lofty, 2,334 feet, is only twelve miles by motor, and can be reached over an excellent bituminous road in thirty minutes. Many of the more wealthy citizens possess summer residences in the ranges, and there are some fine houses and gardens at Mount Lofty, Stirling, Aldgate, Blackwood and other hill resorts. Along the slopes of the mountains, in areas of about twenty-five inch rainfall, lie the principal vineyards. South Australia, with its Mediterranean climate, leads the Commonwealth in wine production, and many vineyards within seventy miles of Adelaide will repay a visit.

Very popular are the seaside resorts on the Southern Ocean, which can be reached over bituminous roads in an hour and a half. Victor Harbour, which was a whaling station in the early days, is beautifully situated amidst the granite cliffs and sandy beaches of Encounter Bay. Near at hand are the lakes of the lower Murray, which provide excellent duck shooting, while the river mouth can be reached by launch from Goolwa, a busy river port in the days when South Australia developed, and for a time held, the steamer trade of the river.

Adelaide is the starting point for the transcontinental journey to Darwin, a most attractive trip through the deserts and empty interior of the continent. No journey gives the traveller so real a knowledge of the red heart of Australia. The route passes successively through fertile wheat lands, well-watered sheep country, saltbush sheep lands, the interior deserts and the cattle country of the far north. The inland regions, with their desert colouring and vast emptiness, have an extraordinary fascination. By rail and motor the continent can be twice traversed—to Darwin and back—in a little more than three weeks. Adelaide and the other great cities of Australia are not unlike the great cities of many other countries. The city people of Australia live lives which are not dissimilar to those of their fellows in the cities of many other countries. The visitor who can spare the time to cross Australia will leave the Commonwealth with a knowledge that few Australians themselves possess—a realization of the magnitude of this young country, an understanding and sympathy with the problems of pioneering, and a true appreciation of the overwhelming difficulties which prevent any nation—white or coloured—from developing the greater part of the continent.

Adelaide and suburbs from the foothills of the Mount Lofty Ranges.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on November 11, 1936, Dr. Walter Summons, the President, in the chair.

Athletics and the Heart.

DR. ERIC COOPER read a paper entitled "Athletics and the Heart". The paper, which had been written by himself, Dr. John O'Sullivan and Mr. E. Hughes, was illustrated by a number of latern slides (see page 569).

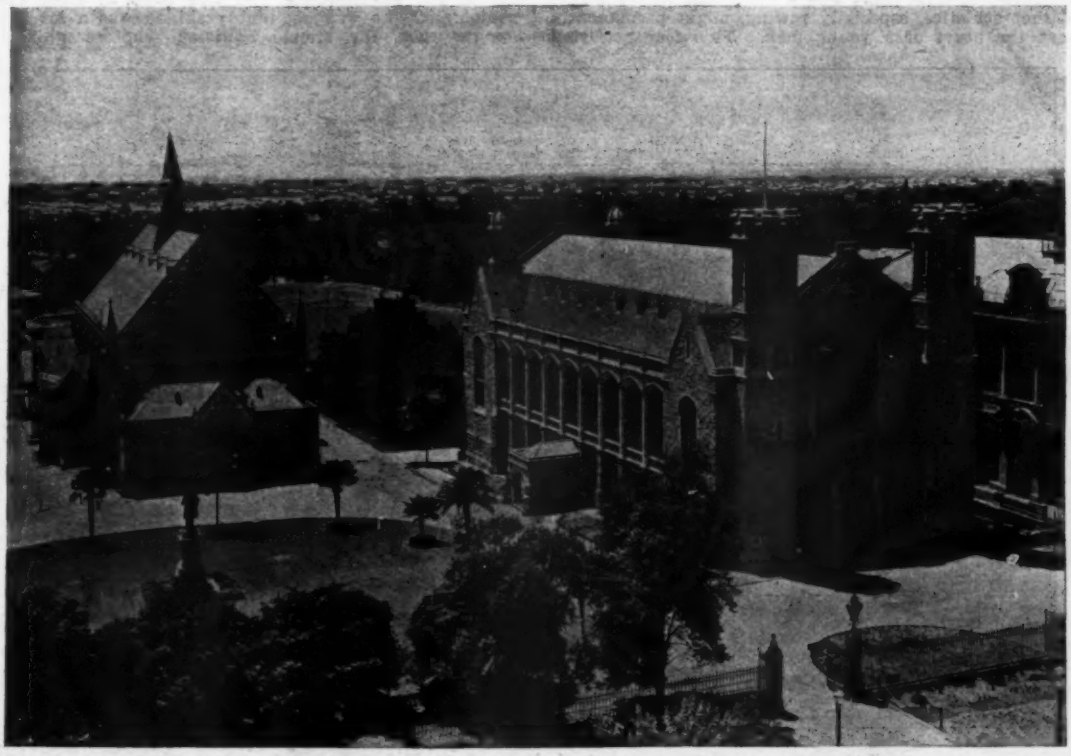
DR. JOHN O'SULLIVAN said that he was speaking because of Dr. Cooper's insistence that he should say a few words about the radiographic side of the investigation. The subject matter of the paper was clinical, and the radiological aspect, although important, was not the chief aspect. Dr. O'Sullivan did not think that a lengthy discussion of the radiological technique, any more than a discussion devoted exclusively to the electrocardiographic side of the paper, would be in keeping with the purpose of the authors or the wishes of the meeting. Dr. Cooper had referred to most of the important points from the radiographic side. There were, however, some on which Dr. O'Sullivan would like to elaborate. The films were made at a six feet distance. The earlier films were made with a rapid exposure technique. The later films were given long exposures, two to three seconds, so that an outline of the heart in diastole should be obtained. In order to obtain a heart outline during actual exercise the subject was placed on the "stationary bicycle" and by means of immobilizers the trunk was firmly fixed to the plate-changing device. In this way it was possible to make films of the heart at rest, during exercise, immediately after stopping exercise, and three minutes after stopping. The slides shown by Dr. Cooper illustrated the importance of taking the films in full inspiration in every case, and illustrated the variations in heart outline with the different phases in respiration. For the purpose of the study they had not used the measurements of the lateral silhouette as seen in the lateral view. This had already been done in a series of cases by Rahlston Patterson, and he had shown that there was a correspondence between the postero-anterior and lateral views. Dr. O'Sullivan concluded by emphasizing the fact that the measurements were not to be taken in a strict mathematical sense, but rather as comparative measurements.

PROFESSOR W. A. OSBORNE said that at the beginning of the twentieth century very little reference to athletics could be found in physiological and medical literature. The case was very different today. Recently a whole issue of the *Deutsche Medizinische Wochenschrift* had been devoted to medicine in sport. The more important physiological contributions to the interesting subject so ably handled by Dr. Cooper might be given as follows.

In the first place, that remarkable reflex arising from the *sinus caroticus* and aortic arch, to which so much attention was paid today, was completely abolished in muscular exercise. There was no "vagal saving" or "depressor" arteriolar dilatation. This had not received the attention it deserved.

In the second place, there was a great difference between exercise with psychic excitement and exercise without it. Thirty years previously the difference had been explained by the superior vascular adjustments through nerve channels when there was rivalry or danger, harking back to primitive fight or hunt. Today they knew that in addition to the nervous factor there was the outpouring of adrenaline, which not only caused a mass transference of the blood from the skin and viscera to the somatic muscles, but also mobilized sugar and probably also potassium, and had some strange direct action on the muscle, probably allowing an increase of oxygen overdraft.

Thirdly, a skeletal muscle much resembled the heart in its blood circulation, which was chiefly diastolic. A



Bonython Hall, North Terrace, Adelaide.

Photograph by courtesy of the South Australian Publicity and Tourist Bureau.

strongly contracted muscle offered great resistance to the arterial flow, though the act of contracting aided the expulsion of venous blood. Fortunately, in most exercises there was reciprocating action, flexion following extension, and *vice versa*. In sustained contraction, as occurred in tug-of-war, the arterial and venous pressures rose and, as pointed out by A. V. Hill, the efficiency dropped.

In the fourth place, training diminished the thickness of the intermediary layer of lymph between contracting fibre and capillary. This not only facilitated gaseous interchange, but reduced the viscous resistance of the muscles, thus lowering the fraction of energy turned into heat.

Dr. Hume Turnbull said that he wished to express his thanks to Dr. Cooper and his associates for their most interesting communication, which had entailed much hard work and ingenuity. It would be very wise if some *résumé* of this work and its results could be given to the public Press, because there was a great fear in the public mind that athletics, especially rowing, might permanently damage the heart of a young man. This fear persisted despite much evidence against it and none for it. Though Dr. Turnbull had seen many persons in whom athletics were supposed to have caused harm, he had never been able to satisfy himself that this was the case. Often a youth tried to continue training while affected by some mild toxæmia, such as a cold, and showed symptoms of faintness or collapse, but there was no permanent damage and reassurance, with a short rest from exertion, was quite sufficient to restore his health; in very many cases training had been successfully resumed soon afterwards.

Dr. Turnbull thought that far too much attention was paid to the possibility of strain in athletics, and he did not believe that any boy, unless he had some obvious cardiac lesion, would harm his heart by games. If he was not able to support the physical effort, the fact would be obvious even to an unintelligent coach, and few coaches were unintelligent. The present custom of directing exaggerated attention to the heart was very bad for boys and undoubtedly made invalids of some quite healthy lads, who, if left alone, would never give a thought to their hearts. In some cases paroxysmal tachycardia caused confusion and was ascribed to athletics, though it was at least as common in the sedentary.

The unusual electrocardiographic findings also should be stressed. Similar though even more marked findings had been recorded before by Bramwell and Ellis and others, a fact which should restrain clinicians when they felt tempted to give great weight to the findings of so-called instruments of precision. It sometimes happened that similar curves, when found in patients suffering from symptoms that might be due to cardiac damage, were regarded as absolute proof of a coronary lesion. It was obvious, from Dr. Cooper's work, that, in the present state of ignorance, dogmatic opinions could not properly be based upon such curves alone. The instrument might be precise, but the interpreter was very fallible. Dr. O'Sullivan had demonstrated the same thing in regard to the X ray examination, and had made it clear that a radiologist who was not highly skilled was a far greater danger than the simple clinician who recognized his limitations. Many adults ascribed their eventual cardiac breakdown to their athletic activities in youth, but Dr. Turnbull had never been able to trace any relationship, and the condition found was always one which was quite frequently seen in the non-athletic. Dr. Cooper's paper had been most interesting and instructive, and it would be a great pity if its lessons were not published widely to the lay as well as the medical public.

The Anatomy School and part of the gardens, University of Adelaide.

By courtesy of the University of Adelaide.

Dr. M. D. Silberberg said that J. B. S. Haldane had stressed the advantage of being one's own rabbit for the purpose of physiological experiment. Dr. Cooper had followed this excellent advice, and in addition had made full use of the willingness of the Ormond crew to cooperate in his investigations on athletes and the heart. This was in the best tradition of clinical science and the modern approach to physiological problems.

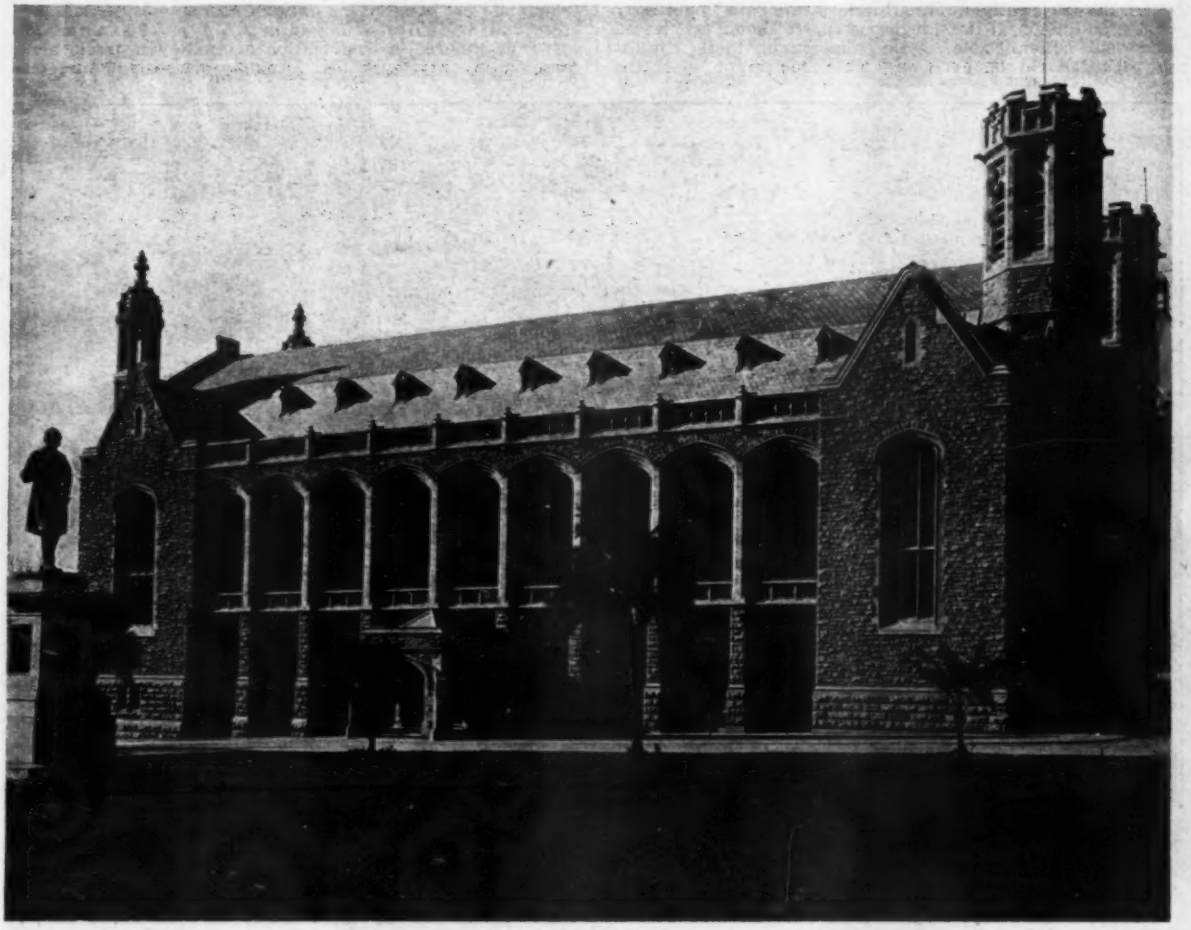
One valuable feature of his paper was that he demonstrated that increase of the size of the heart was not synonymous with pathological dilatation or even physiological hypertrophy, but that the enlargement was due to

increased diastolic filling and was not to be regarded as a serious sign. He and Dr. O'Sullivan had confirmed the work of others that during strenuous exertion in trained men the heart became smaller and not larger, contrary to the view held by many medical men and the laity, with especial reference to the legal fraternity.

If the results set forth in the papers were widely known, there would be much less unnecessary fear of strained heart in athletics. In consequence there would be fewer cardiopaths and less restrictions interfering with the career, livelihood and happiness of a considerable number of young people, diagnosed incorrectly as suffering from "strained heart" or "athletic heart". This raised the important question: was there such an entity as primary cardiac overstrain; in other words, strain in a healthy heart? Could healthy young males or females suffer from heart strain through strenuous athletics?

Modern authorities denied the possibility, and the investigations of Dr. Cooper and Dr. O'Sullivan supported this denial. The somatic muscles became exhausted before the cardiac muscle, because the heart was delivering its maximum blood supply and the metabolic demands could not be further extended, hence the effort was slowed down or terminated by exhaustion of the neuro-chemical muscular mechanism. The practical difficulty was to know when the heart was healthy. In the majority of cases, after investigation of the past history and full examination, the probabilities of normality were so strong that dogmatism regarding physical fitness was usually justifiable and was justified by the after-results.

There remained a few cases in which atheroma of the coronary arteries or a quiescent toxic focus eluded the most refined means of diagnosis. Dr. Silberberg illustrated this point by a case of his own. A well-known athlete had died at the age of forty-eight from coronary thrombosis supervening on coronary atheroma in the left descending branch. Atheroma was present in the other coronary branches. This was demonstrated at autopsy by Dr. Wright Smith. The left ventricle was enlarged. The rest of the organs were normal. The fatal attack had developed during a day of hurry and rush of work. This man's habits had been impeccable. He was a teetotaller and non-smoker, and took a keen interest in matters of health. For many years he had indulged in weight-lifting, wrestling, running and physical culture. He had been medically examined at intervals of two to four years over a period of seventeen years. Nothing abnormal was discovered, except albuminuria, which was inconstant. The apex beat was from four and a quarter inches to four and three-quarter inches from the mid-line. His systolic blood pressure was from 120 to 130 millimetres of mercury and the diastolic pressure 80 millimetres. The patient's electrocardiograms had been normal at the date of Dr. Silberberg's last examination eighteen



The Bonython Hall, University of Adelaide.

By courtesy of the University of Adelaide.

months prior to the patient's death. It was likely that the atheroma was of long standing, yet there had been no signs or symptoms of its presence, at least up till eighteen months of death.

In another case, a professional wrestler, aged thirty-six years, in 1927 had had a typical attack of coronary thrombosis. He had a pericardial rub, lasting twenty-four hours, and an electrocardiogram taken six days later had shown deep inversion of *T* in Lead I. He had made an apparently complete recovery and participated in wrestling matches for several years; since then Dr. Silberberg had not heard of him. Almost certainly in such a case coronary atheroma in a small branch was the basis of the attack, yet apparently the patient had had no previous symptoms or signs.

In the physically unfit, athletic strain was manifestly risky, and amongst the dangerous sports should be classed the "old buffers' race". But that night these clinical by-paths should be avoided. The problem of sport for schoolboys and exercise in middle and old age was also somewhat outside the scope of this discussion.

The Darling Building, University of Adelaide.

By courtesy of the University of Adelaide.

The question of toxæmia in athletes was one of importance. In severe toxæmia, such as occurred in acute tonsillitis or influenza, strenuous exertion could not be indulged in. In the mild grades of toxæmia, such as occurred in coryza, pharyngitis, mild sinusitis, slight diarrhœa or even fibrositis, the individual felt out of sorts and listless, but not ill enough to be forced to bed. He was loath to let down his team-mates, to disappoint the public or to waste the weeks or months of training. Yet in these circumstances, not only would his effort be a poor one, well below his true form, but also there was a definite possibility of producing the condition known as effort syndrome. Strictly speaking, this was not primary cardiac over-strain, because the heart muscle was temporarily involved in a toxic state, even if only to a mild degree.

Effort syndrome was often incorrectly regarded as due to myocarditis. The tachycardia resulted from disturbance of the neuro-chemical mechanism. The athlete, in spite of his mild toxæmia, insisted on taking part in strenuous sport. Driven by his will power, the urge to succeed and the discipline and tradition of team work, he strove on till more or less collapse was induced. Dyspnœa was marked. Tachycardia persisted and exhaustion was extreme. Syncope might occur, and precordial distress or pain was not unusual. This condition might clear up after some days, but on the contrary it might persist for weeks or months. The heart might be somewhat enlarged and a functional bruit might be present. In these circumstances a diagnosis of "strained heart" or "leaking valve" would add further anxiety to the apprehension already possessed by the patient as a result of subjective symptoms of palpitation, fatigue, shortness of breath, precordial discomfort and faintness. A psychotoxic state was thus induced, a combination of anxiety neurosis and toxæmia in varying ratios. Anxiety neurosis acting on the autonomic nervous system and on the endocrines might cause persistence of tachycardia, nervousness, tremors, sweating *et cetera*.

Anxiety neurosis was an interesting condition. If they analysed the symptoms of fright, the anxiety syndrome was revealed, less intense, but more persistent. Medical practitioners should be careful not to create this type of anxiety neurosis. Mackenzie, with justice, referred to "doctor-made neurasthenics".

Turning to another aspect of athletics and heart strain, what should be their atitude to hyperpiesia in youth? For example, a prominent league footballer might be found to have a persistently raised pressure of about 160 millimetres. A psychical rise of pressure was excluded; there might be a mild grade of arterial sclerosis without evidence of renal impairment, and the family history was good. One could not be absolutely sure that patchy atheroma of coronary or cerebral arteries was absent, and in theory at least they should play for safety and advise against strenuous athletics. Actually the risk was prob-

ably slight, and in some at least of these cases the high systolic pressure might be merely the expression of a relatively slow heart rate, an increased filling during diastole, and therefore a large output with the peripheral arteries insufficiently relaxed.

That evening they had listened to the results of certain measurements of pulse rate, blood pressure, respiration rate, the size of the heart and the electrical changes. These methods were available to all clinicians and were but simple working tools. With these and evaluation of symptoms they based opinions of health and disease. But when these observations were analysed physiologically, the complexity of the neuro-muscular chemistry and physics was only too apparent. The knowledge of the chemical mediation of the nervous impulse was comparatively recent, and who could doubt that much more remained to be discovered in the complex chemistry of cell activity and the harmonizing of the multitudinous reflexes and interactions which occurred in the human organism.

The fact that certain record-breaking animals had unusually large hearts was not a conclusive argument that strain had caused the enlargement. It might with justice be contended that they were star performers because they possessed unusually powerful hearts.

In estimating the size of hearts, allowance must be made for build. A tall individual had a hanging type of heart, whereas a broadly-built, sthenic person had a heart more transversely placed. Enlargement in the hanging type of heart was much more difficult to assess than in the broad heart. Allowance should be made for this fact in comparing the hearts of, for example, sprint runners of the tall type, and rowers, wrestlers and others of the stocky, broad build with deep chests. The same remarks applied no doubt to comparative studies in the size of animals' hearts.

In conclusion he agreed with those who maintained that primary cardiac strain was uncommon, that "athlete's heart" in a pathological sense was a rarity, and that the longevity of athletes was not shortened because of physical strain in earlier life.

DR. J. F. MACKEDDIE said that such a paper was all too rare; it contained a report of a piece of clinical research work of a very high order. Speaking as an old physician looking back, he wished to draw attention to the extraordinary advances that had been made in cardiology; instead of merely tracing and placing murmurs, the very means of measuring cardiac efficiency had been evolved. It had been established that the heart was a very good organ, the resistance of which was great and the vulnerability high. He deprecated the habit of nursing hearts and making invalids of people with something to hear near the heart. He recalled the case of a lad of fourteen, with a roaring systolic murmur, breathlessness and cardiac failure, who was expected to become an invalid. By means of regular systematic exercises and rare determination to overcome his physical handicap, this same lad had been seen by Dr. Mackeddie again at the age of twenty-five years because of a temporary stiffness of the knee. He had become a champion harrier, who could run ten miles with ease, and his pet hobby was the felling of trees. There was something very staunch about the heart as a system of muscular chambers fitted with valves. The question of vascular lesions, however, was quite a different thing. The onset of atheroma, for example, was very insidious, and such a condition was likely to be progressive. Dr. Mackeddie thought that perhaps he might comment on the work reported by Dr. Cooper by drawing attention to the lack of correlation between

The Johnson Chemical Laboratory, University of Adelaide.

By courtesy of the University of Adelaide.

changes in the size of the heart and the electrocardiographic tracings and the records of rise and fall of blood pressure. The subject was very complex and difficult to grasp; it was hard to determine where the vulnerability of the heart came in, other than the vascular part. In conclusion, Dr. Mackeddie said that surely it was a duty to make it known widely that "strained heart" was a figment of the imagination.

Dr. W. W. Ostermeyer expressed his appreciation of the paper and emphasized the prime importance of discriminating between exertions comprising alternating flexion and extension, and exertions requiring prolonged subjective intensity of strain, by recounting some of his personal recollections of tugs-of-war and wrestling. He described wrestling as a deadly business, and considered that the choking associated with such a grip as the half-Nelson would certainly cause heart strain. He dissented against the idea of giving up strain of cardiac muscle as an entity. He also drew attention to the necessity for considering the degree of rotation of the heart when skiagrams of the chest were used for estimating the size of the heart.

Dr. Cooper, in reply, said that he was glad that Professor Osborne had stressed the importance of the disappearance of vagal saving during exercise. In the paper it was pointed out that the vagotonic state of the athlete at rest became one of extreme sympatheticotonia during exercise. So far as their observations went there was no evidence that exercise even of severe grade could harm the untrained heart.

In reply to Dr. Turnbull, Dr. Cooper admitted the importance of "doctor-made" cardiacs. No man who broke down during training should have the heart mentioned when his condition was discussed. A general state of staleness, a general toxæmia should be the explanation rather than any possible local effect on the heart. Breakdown during training was rarely, if ever, due to cardiac causes.

In reply to Dr. Silberberg, Dr. Cooper said that he regretted that his statement, that abnormal hearts did not dilate after exertion, had been misinterpreted. Although there was no evidence from their experiments that damage was done to abnormal hearts by exercise, they had not sufficient facts to go on to extend the statement "that exercise could not harm an abnormal heart".

In reply to Dr. Mackeddie, Dr. Cooper said that a correlation had been attempted between the various observations made, and there seemed to be no simple relationship between changes in heart size and the electrocardiogram during training or exercise. The reduction of clinical facts to a numerical formula was not the object of clinical medicine. Exercise and other tests for physical fitness had been evolved, but the majority of good athletes would fail to pass these rigid limitations.

In reply to Dr. Ostermeyer, Dr. Cooper said that the factor of scoliosis was well known in radiology and had been common in the students examined. Many films had to be rejected because of this factor.

In conclusion, Dr. Cooper thanked the speakers for their assistance and pointed out that this was merely the commencement of a line of research, which it was hoped would develop in the course of the next few years.

NOMINATIONS AND ELECTIONS.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Johnston, George Arthur William, M.B., B.S., 1937 (Univ. Sydney), Sydney Hospital, Sydney.

Pro-Copis, George Sandford, M.B., 1936 (Univ. Sydney), Sydney Hospital, Sydney.

Macfarlane, John Stuart, M.B., 1908 (Univ. Sydney), 35, Main Street, Lithgow.

Correspondence.

GASTRIC ULCER: INTERPRETATION OF CERTAIN RADIOGRAPHIC APPEARANCES.

Sir: There is an alternative explanation for the radiographic changes interpreted as "penetrating ulcer" and its "cure" as used by Dr. John H. Halliday in reporting a case of gastric ulcer in the journal of March 27, 1937.

For some years radiologists have commented on the dramatic and rapid disappearance of large ulcer niches in certain of these patients with different forms of treatment. Barclay (*The British Medical Journal*, December 8, 1928, page 1026) has discussed this matter and offered an explanation for the radiographic changes, which is now generally accepted.

I have taken tracings of the lesser curvature of the stomach from the films accompanying Dr. Halliday's report for the purpose of demonstrating the probable pathological alterations responsible for the radiographic picture.

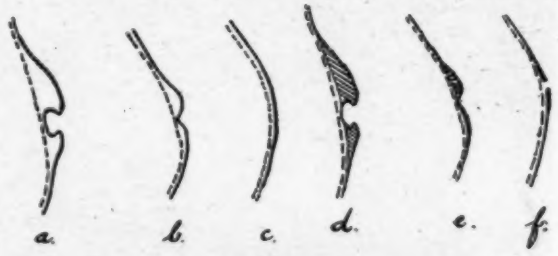


Figure I.

In Figure I, *a*, *b* and *c* are tracings from the three radiographs. The dotted line is added to suggest the muscle layer of the stomach. *d*, *e* and *f* are the same tracings with cross-hatched areas to suggest heaped-up mucosa in *d* and *e*, and a break in the continuity of the apparently normal flattened mucosa in *f*.

A study of Barclay's paper will show the details connected with the sequence of changes depicted. It seems, therefore: (i) that care is required before the diagnosis of "penetrating ulcer" is made from certain radiographic appearances; (ii) that the radiographic examination may not reveal the mucosal ulcer if the surrounding membrane is not swollen. It is likely that the condition of affairs shown in *f* explains the 5% to 15% of symptom-producing gastric ulcers that escape radiological detection. There are many pitfalls to be avoided in using radiological findings as standards of cure of these lesions.

Yours, etc.,

F. L. Gwynne.

3, Alfred Street,
Auckland,
New Zealand.

April 1, 1937.

Obituary.

STANLEY ROSSITER BENEDICT.

We are indebted to Dr. H. S. H. Wardlaw for the following appreciation of the work of the late Stanley Rossiter Benedict.

America has lost heavily of her pioneers in biological chemistry during the last two years. Otto Folin, of Harvard, and Lafayette B. Mendel, of Yale, have died during this period. News is now to hand of the death of Stanley R. Benedict, of Cornell, at the age of fifty-two.

Since 1910 S. R. Benedict has taught biological chemistry in Cornell University Medical College. His death, following so closely on that of Folin, closes a long period of rivalry

and fruitful controversy between the schools of biological chemistry at Harvard and Cornell. Much work on similar lines was done at each of these schools, but the points of view of their heads were very different. To both schools we owe numerous methods for the analysis of blood and of urine.

Folin was always rather impatient of minutiæ. He frequently arrived at the essentials of his problems with an insight and economy of means which were astonishing. In Benedict, on the other hand, there was a tendency to emphasize detail and to stress the exceptional. He refused to accept any conclusion of which he did not approve as long as the slightest shred of evidence against any part of it remained. His attitude in the long controversy with various workers on the creatinine of the blood was typical of this. For a time he denied that creatinine was even present in the blood as such. When the weight of evidence became overwhelmingly in favour of its presence, he still insisted that the data presented did not prove that the whole of the material estimated as creatinine was this substance. He was still actively working on this problem at the time of his death.

S. R. Benedict's name is probably best known through the almost universal use of his reagent for the detection of reducing sugar in urine. It is interesting to notice that he devised this reagent while a second-year student. Some idea of his energy and enterprise as a student may be gathered from the fact that he published nine original papers before graduation, besides assisting in the teaching of chemistry in his university (Cincinnati). He studied medicine for a short time, but abandoned this to devote himself entirely to chemical work.

Benedict's most important work is probably the series of investigations by which he demonstrated the exclusive formation of urinary ammonia by the kidney, apart from any that might be absorbed from the alimentary tract. He showed the importance of this mechanism for the maintenance of normal tissue reaction, and the correlation of its failure with the onset of vomiting in advanced stages of renal deficiency.

For the past ten years Benedict had done valuable service for biological chemistry in carrying out the exacting duties of managing editor of *The Journal of Biological Chemistry*. His loss will be severely felt in many ways.

A SKIT.

A CORRESPONDENT who wishes to be anonymous has forwarded the following skit on "Current Comment".

In these days of technical medicine it is probably true, even if regrettable, that more interest is taken in advances of a partly technical kind than in those concerned with the fundamental mechanism of the body. We are, as Lord Dawson recently said, "gadget-minded". This mild reproof need not concern us when we come to work of outstanding merit and value, such as that published in a recent issue of *The Journal of Cynical Medicine* on an apparatus for spraying the nasal passages of mice. No doubt many readers of these pages must have wished that some simple appliance was readily available for the purpose. Readers will recollect the extraordinary epidemic of a severe fungus infection of the scalp which spread over the rural districts of the eastern part of Australia during the war years. This was due to a remarkable plague of field mice, which invaded towns and dwellings alike, respecting neither the belongings nor persons of the people. Numbers of children were bitten during sleep; in one case it actually happened that the ear of a bank manager was bitten; this must be very rare. In another case it was reported that the ear of a bookmaker was also bitten, but this is almost certainly incorrect. However, a great need was felt at the time for reliable means for trapping the mice, which did not seem to be attracted readily by the usual sapid baits. Had some means existed at the time for spraying their nasal passages so as to remove any congestion or relieve local inflammation, no doubt the increase in acuity of the olfactory sense would have resulted in a much larger harvest in the mouse traps. The device seems to be very simple: manual control of the mouse has been eliminated, so long a trouble in laboratories with female technicians, though a certain periodicity in this variety of mouse-shyness has been noted from year to year, no doubt associated with fluctuations of fashions. The author is perhaps not quite exact in his methods, as he does not seem to have provided for spraying of each nostril singly, nor is there any allowance for deviation of the nasal septum or hypertrophy of the turbinates. This, however, is a detail, and probably only concerns the rhinologist with a "society" practice, where he may be unable, for motives of expediency, to refuse attendance on white mice belonging to the cadets of noble and/or wealthy houses.

Further extension of this interesting work is to be expected, since it was discovered last year that an endemic granuloma in the ethmoid sinus of the dog flea was common in large cities. This gives rise to repeated sneezing on the part of the insect, which is not only a great source of annoyance to the smaller type of long-haired dogs, but is responsible for the setting free of small particles of dog hair through scratching. To this is due the greater incidence of asthma in some modern flats, and the clinician, being naturally interested in prophylaxis, would welcome the elaboration of a nasal spray suitable for the dog flea. This would prevent these troublesome attacks and no doubt save him (the clinician) much valuable time which could be better spent, for if a busy doctor must spend his scanty leisure in opening bottles, these need not always contain adrenaline.

PORTRAIT OF PROFESSOR W. A. OSBORNE.

IT is the desire of the many friends and old students of Professor Osborne that his portrait should be painted and presented to him, and a commission has been placed with Mr. McInnes. A subscription list has been opened, the subscriptions being fixed at half a guinea. Any friends and old students wishing to be associated with this recognition of Professor Osborne's long services to the University of Melbourne are invited to address cheques to one of the undersigned, Medical Society Hall, 426, Albert Street, East Melbourne, C.2.

B. T. ZWAR.
H. B. DEVINE.
ALAN NEWTON.
VICTOR HURLEY.
SYDNEY SEWELL.

Additional subscriptions have been received from the following: H. W. Wunderley, C. Craig, Sara Gunderson, R. H. Morrison (£1 1s.), D. Bird.

The total amount received is now £105 10s. 6d.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1925 to 1933*, of Queensland, as duly qualified medical practitioners:

Bayley, Arthur Wellesley, M.B., B.S., 1923 (Univ. Melbourne), Nambour.
Franklin, Samuel de Vere, M.B., Ch.M., 1921 (Univ. Sydney), Brisbane.
Hooper, Kenneth Hugh, M.B., B.S., 1936 (Univ. Sydney), Brisbane.
Innes, James Ross, M.B., Ch.B., 1927, M.D., 1934 (Edinburgh), D.T.M., 1934 (Liverpool), Brisbane.
Kelly, Ronald William Grant, M.B., 1929, B.S., 1933 (Univ., Sydney), F.R.C.S., 1934 (Edinburgh).
Scott, John Linsley Dalkeith, M.B., B.S., 1936 (Univ. Sydney), Toowoomba.
Yoffa, David Leslie, M.B., B.S., 1918 (Univ. Melbourne), Brisbane.

Books Received.

A TEXT-BOOK OF MEDICAL BACTERIOLOGY, by R. W. Fairbrother, D.Sc., M.D., M.R.C.P.; 1937. London: William Heinemann (Medical Books) Limited. Medium 8vo, pp. 445, with illustrations. Price: 15s. net.

SYDNEY HOSPITAL PHARMACOPŒIA AND REFERENCE HANDBOOK; Ninth Edition; 1937. Australia: Angus and Robertson Limited. Demy 16mo, pp. 113. Price: 4s. 6d. net.

SURGICAL ANATOMY, by G. Massie, M.B., M.S., F.R.C.S.; Third Edition; 1937. London: J. and A. Churchill Limited. Medium 8vo, pp. 478, with 153 illustrations, many of which are in colour. Price: 18s. net.

ELEMENTS OF ORTHOPÆDIC SURGERY, by N. R. Smith, M.B., Ch.M., F.R.C.S., with a foreword by R. C. Elmslie, O.B.E., M.S., F.R.C.S.; 1937. Bristol: John Wright and Sons Limited. Crown 8vo, pp. 257, with illustrations. Price: 10s. 6d. net.

Diary for the Month.

Apr. 20.—New South Wales Branch, B.M.A.: Ethics Committee.
Apr. 21.—Western Australian Branch, B.M.A.: Branch.
Apr. 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
Apr. 23.—Queensland Branch, B.M.A.: Council.
Apr. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Apr. 28.—Victorian Branch, B.M.A.: Council.
Apr. 29.—New South Wales Branch, B.M.A.: Branch.
Apr. 29.—South Australian Branch, B.M.A.: Branch.
May 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
May 5.—Victorian Branch, B.M.A.: Branch.
May 5.—Western Australian Branch, B.M.A.: Council.
May 6.—South Australian Branch, B.M.A.: Council.
May 7.—Queensland Branch, B.M.A.: Branch.
May 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
May 14.—Queensland Branch, B.M.A.: Council.
May 18.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments.

Dr. R. J. Millard has been appointed a member of the Nurses' Registration Board of New South Wales.

* * *

Dr. G. M. Whish has been appointed Government Medical Officer at Young, New South Wales.

* * *

Dr. T. J. Beresford has been appointed Resident Medical Officer at the Adelaide Hospital, Adelaide.

* * *

Dr. A. W. Raymond has been appointed Medical Inspector of Seamen at Brisbane, pursuant to the provisions of the *Navigation Act*, 1912-1935.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii to xxi.

Hobart General Hospital, Hobart, Tasmania: Resident Medical Officer.
Lewisham Hospital, Lewisham, New South Wales: Honorary Radio-Therapeutist.
Public Service Board, Adelaide, South Australia: Assistant Bacteriologist.
Repatriation Commission: Resident Medical Officer, Caulfield, Victoria.
Renwick Hospital for Infants, Summer Hill, New South Wales: Honorary Assistant Ophthalmic Surgeon.
Royal Hospital for Women, Paddington, New South Wales: Honorary Officers.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
South Australian: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.